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A.M.A. ARCHIVES OF DERMATOLOGY

The Interrelation of Some Cutaneous and Ocular Diseases
Joseph V. Klauder

Senile Purpura and Liver Disease
Vincent J. Derbes and Marvin E. Chernosky

Colloid Degeneration of the Skin (Colloid Milium)
Jere D. Guin and Everett R. Seale

Incidence of Dermatophytes in Cincinnati and Some Diagnostic Laboratory Procedures
J. Schwarz

Cholinesterases in Human Skin
R. K. Winkelmann and Robert W. Schmit

Fox-Fordyce Disease
Leopold F. Montes, Alonso Cortes, Burton L. Baker, and Arthur C. Curtis

Specific Systemic Antibiotic Therapy in Familial Benign Chronic Pemphigus
Walter B. Shelley and Donald M. Pillsbury

Visual Disturbances with Antimalarial Drugs, with Particular Reference to Chloroquine Keratopathy
H. E. Hobbs and C. D. Calnan

Unusual Eruptions Associated with Internal Malignant Disease
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Postfuruncular Alopecia
Thomas Butterworth and John C. Fowler

Report: Progress in Biological Sciences in Relation to Dermatology
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Report: IV Congressos de Dermatologia Ibero Latino-Americano e Luso-Espanhol
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Adaptation of the Safety-Pin for Use as a Desiccating Needle
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Wart Relapses at the Edges of Therapeutic Cantharidin Blisters
George H. Findlay

Erythema Gyratum Repens
M. J. Purdy

Correspondence

Obituary: Erich Hoffmann, M.D.

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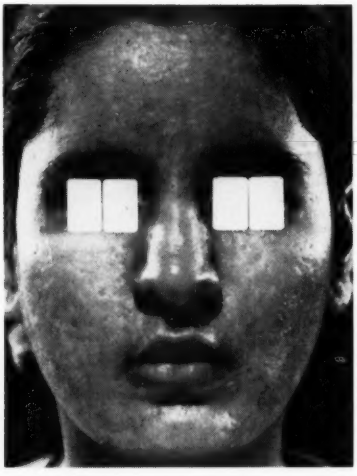

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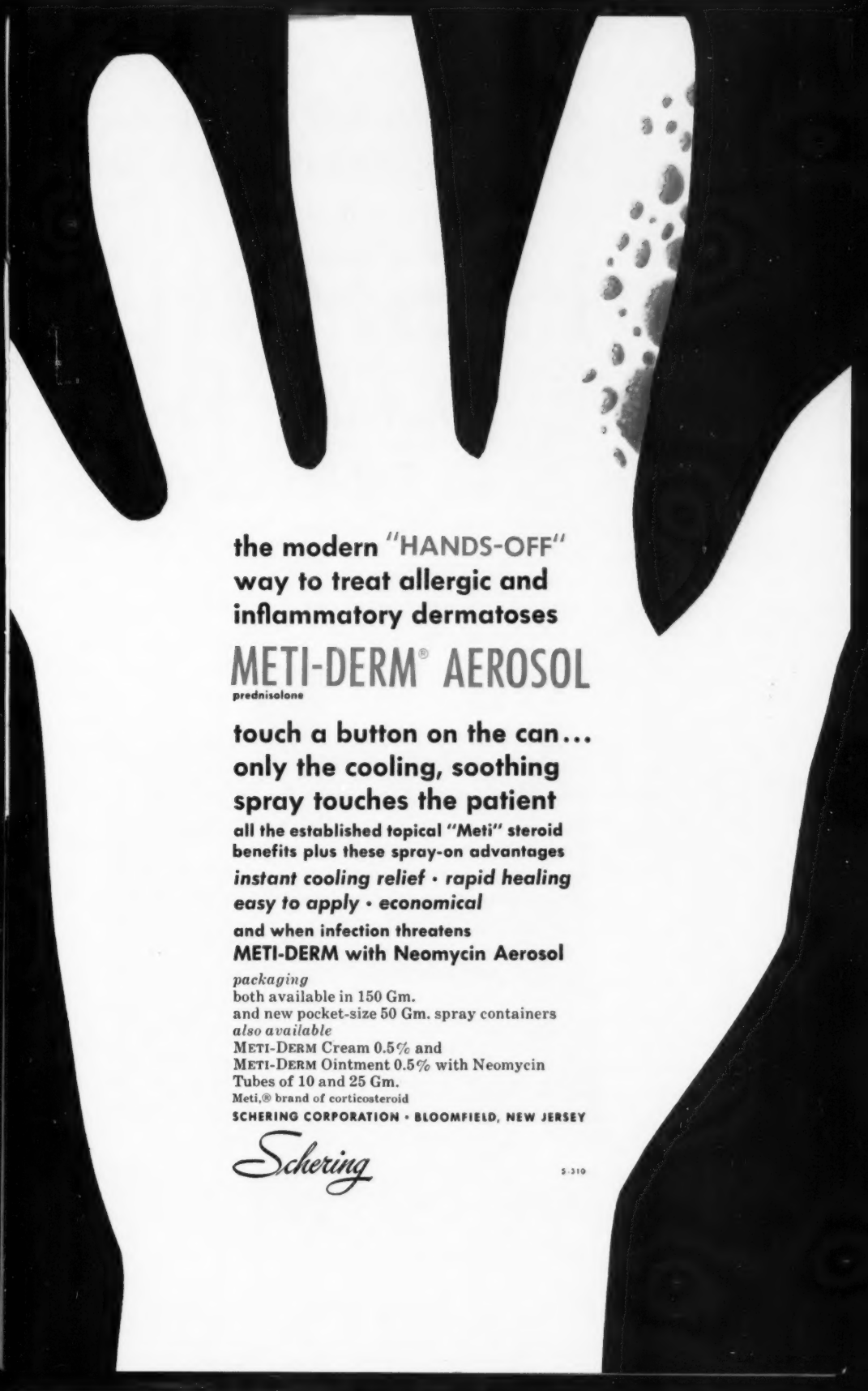

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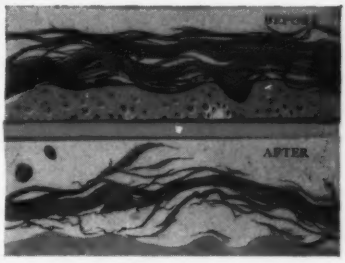

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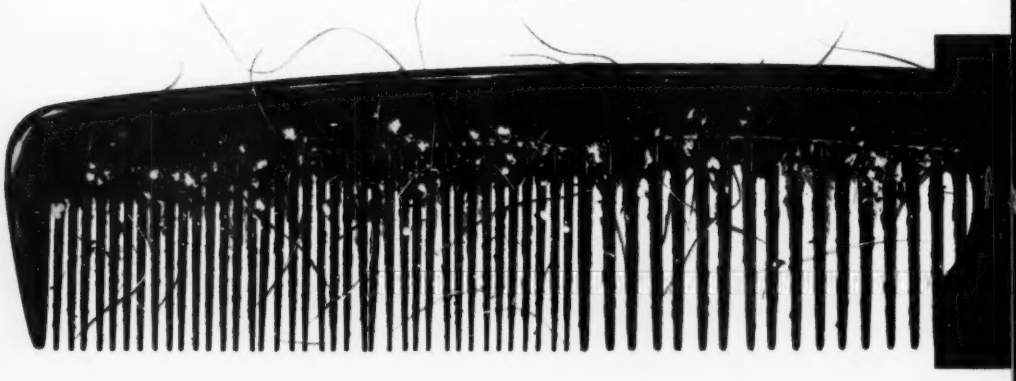

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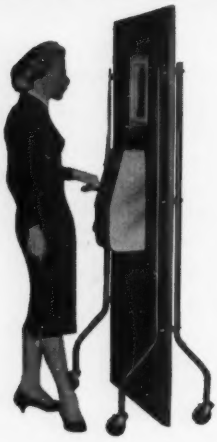

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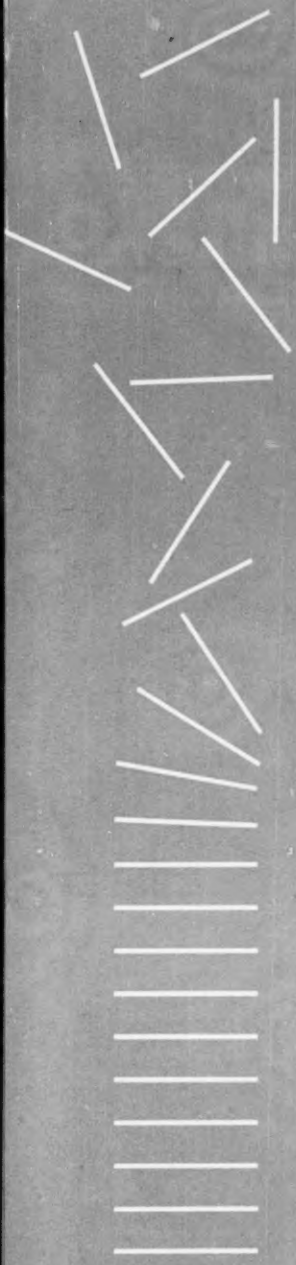

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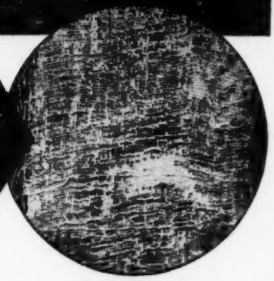

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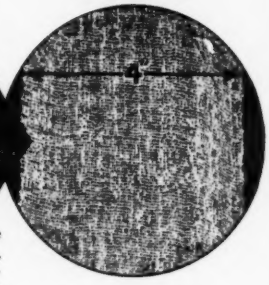

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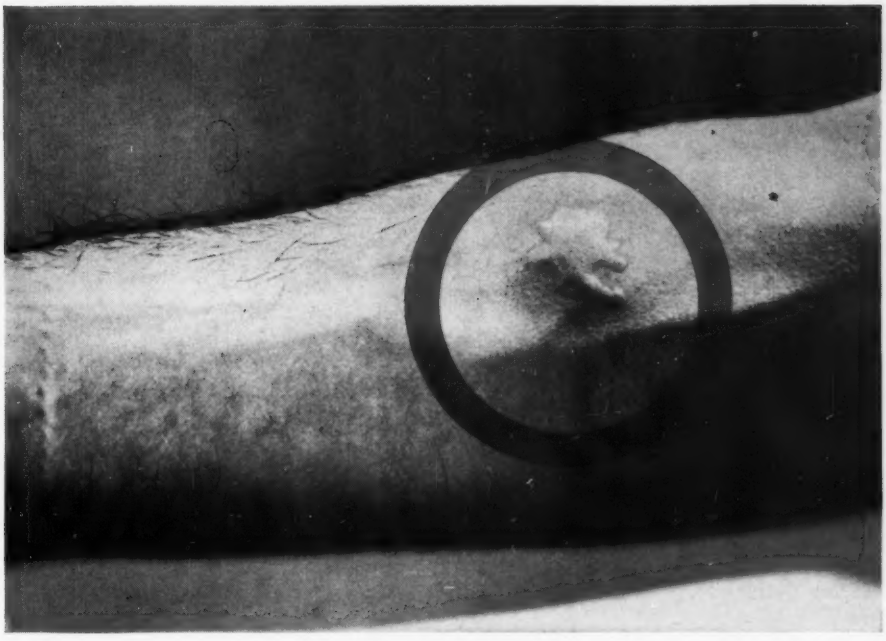

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2. R. C. V. Robinson and L. E. Harmon, *Antibiotics Annual 1958-1959*, New York, Medical Encyclopedia, Inc., 1959, p. 113.
3. Reports on file, Roche Laboratories.
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5. F. T. Becker and J. L. Tuura, paper read at Conference on Recent Contributions to Antibacterial Therapy, New York City, May 21-22, 1959.
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TRIBURON® Chloride — brand of triclobisonium chloride — N, N'-bis [1-methyl-3-(2,2,6-trimethylcyclohexyl) propyl]-N, N'-dimethyl-1, 6-hexanediamine bis (methochloride)

"severe decubitus ulcers...healed"*

"...a paraplegic had severe decubitus ulcers [which had been resistant to previous treatment]. With PANAFIL ointment...the sacral decubitus ulcer was healed and there was marked improvement in the condition of the hip....No surgical intervention was necessary."—Burke, J. F., and Golden, T.: Am. J. Surg. 95:828, 1958.

panafil® topical enzyme therapy

- effective wound cleansing and healing
- economical for hospital and patient
- well tolerated and convenient for outpatient use

Rystan
Mount Vernon, N.Y.

PANAFIL ointment (papain—urea—water-soluble chlorophyll derivatives). For complete information see your Physicians' Desk Reference

33189

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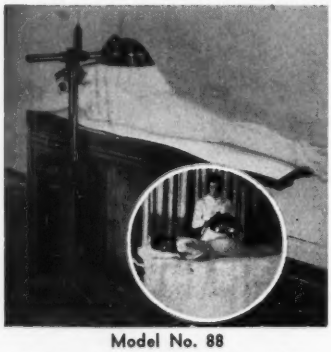

Model No. 88
Fischer Dry Pads For Low Volt
No Moisture * Send For Details

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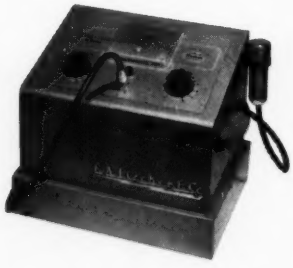

RF-7 Ultra Sound with new 6 or 12 foot very thin, flexible low volt cables.

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BASIC
IN ANY SUCCESSFUL ACNE PROGRAM:
SCRUPULOUS CLEANSING OF THE SKIN

ACNE-DOME®

CLEANSER pH 4.2
U.S. Pat. No. 2,876,164

medicated cleansing cream for skin and scalp in acne

multiple action—thorough but gentle

- restores normal skin acidity to aid healing, prevent further infection
- produces a sea of suds . . . without harsh soaps or detergents
- leaves skin clean and dry . . . but not too dry
- maintains optimal skin-peeling . . . just enough to help remove comedones

Contains bithionol 1%, sulfur 2%, salicylic acid 2% and Colloidal-Soy-Bean-Fraction in the exclusive ACID MANTLE® (buffered aluminum acetate) vehicle.

Supply: 4-ounce jars.

* A cellulose sponge accompanies each jar for more complete, more effective application of ACNE-DOME Cleanser.

DOME CHEMICALS INC. 125 West End Ave./New York 23, N. Y.
Los Angeles / Montreal • *World Leader in Dermatologicals*

A PHYSIOLOGIC WET DRESSING FOR SKIN DISORDERS

PROPHYLLIN®

POWDER FOR WET DRESSINGS

PROPHYLLIN combines sodium propionate, a constituent of the protective secretion of normal skin, with water-soluble chlorophyllin. The result is a **natural,** nontoxic wet dressing, soothing in the most acutely inflamed skin disorders. It can be used even around the eyes and genitalia—or as a mouthwash—without fear of irritating the mucous membranes.

PHYSIOLOGIC: contains no chemical irritants or sensitizing agents

NONASTRINGENT: will not block sweat ducts

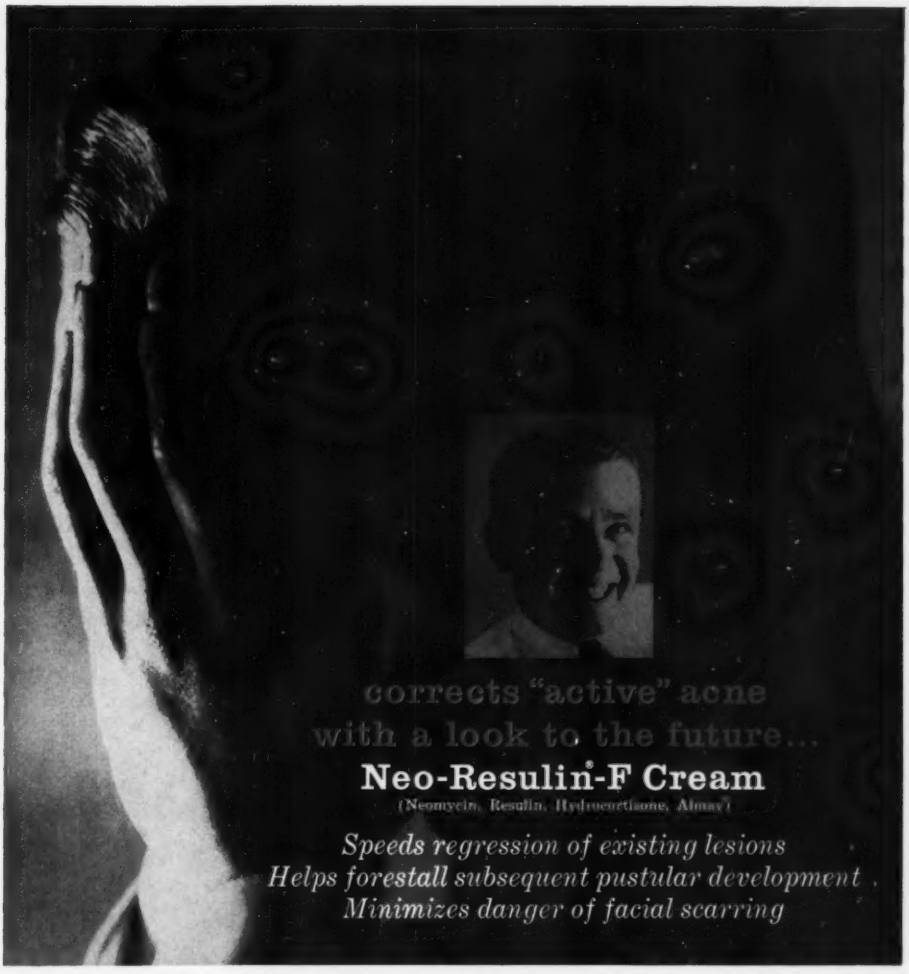
MARKEDLY ANTIPRURITIC

MILDLY BACTERIOSTATIC AND FUNGISTATIC

COSMETICALLY ACCEPTABLE: no staining, no objectionable odor

SUPPLIED:
In cartons of 12 packets. (Each packet contains 2.3 gm. of powder, for preparation of 8 fluid ounces of solution containing 1% sodium propionate and 0.0025% water-soluble chlorophyllin.) Also in 4-ounce and 16-ounce jars.

Rystan company MOUNT VERNON, NEW YORK


corrects "active" acne
with a look to the future...
Neo-Resulin-F Cream
(Neomycin, Resulin, Hydrocortisone, Almay)
Speeds regression of existing lesions
Helps forestall subsequent pustular development
Minimizes danger of facial scarring

New multifaceted Neo-Resulin-F provides unusually broad corrective and prophylactic action in acne rosacea and acne vulgaris. Resorcin and sulfur supply basic keratolytic, antiseborrheic, dehydrative action. Hydrocortisone controls inflammation, hyperemia and pruritus and has been postulated to forestall hyperkeratosis at follicular openings, thus preventing subsequent occlusion. Neomycin reduces bacterial growth and clears pustular infection, thus limiting the possibility of permanent scarring.

If a topical antibiotic is not desired, Resulin-F is available without Neomycin.

Composition: Neomycin Sulfate 0.5% (equivalent to 0.35% Neomycin base); Resorcinol Monoacetate 1.5%; sulfur 2.0%; Hydrocortisone 0.5%; Alcohol by weight 8.5%. *Dosage:* Apply 3 times daily to lesions and associated skin areas. *Supplied:* 15 Gm. tubes. *Samples and Literature Available on Request*

ALMAY Division of *Schieffelin & Co.* / *Since 1794* New York 3, New York

*TELES

Torch (available on prescription in 4 oz. bottles)

Really new, prompt and effective therapeutic agent in control of seborrheic dermatitis, itchy scalp and dandruff.

TELES • is a milk-white, unscented shampoo
TELES • may be employed at weekly intervals or less frequently
TELES • does not stain hair of any color
TELES • does not stimulate oiliness in the scalp
TELES • complete directions for use on each 4 oz. bottle

Samples and literature on request

Reference: Gross E. R. and Wright C. S., A. M. A. Archives of Dermatology, 78:92, July, 1958

TORCH LABORATORIES, INC.

110 Chestnut Street • Philadelphia 6 • Pa. *Tellurium Dioxide Suspension, Torch

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normal protective cover

Normal skin has an acid pH range between 4 and 6

Samples and literature on request

Supply:
ACID MANTLE Creme: 1 oz. tubes, 4 oz. & 16 oz. jars.
ACID MANTLE Lotion: 1 oz. and 4 oz. squeeze bottles, 1 pint bottles.

ACID MANTLE®

CREME or LOTION pH 4.2

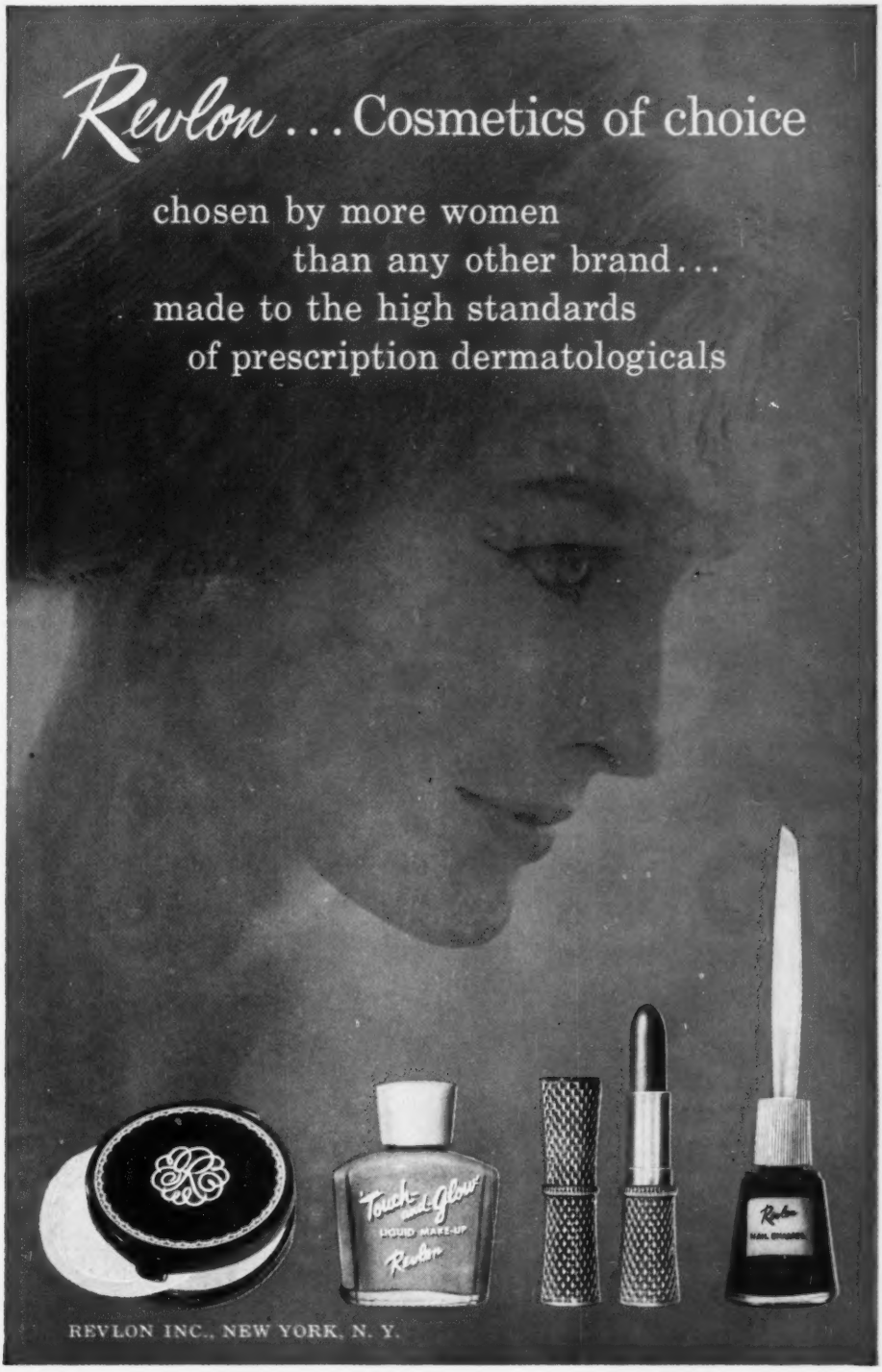
In housewives' eczema and industrial dermatitis, promptly restores and maintains the normal protective acidity of the skin. Active ingredient is aluminum acetate in specially formulated bases.

"The importance of a measure designed to return the pH of the skin quickly to its normal values after exposure to soap and alkalies is amply borne out..."

—Gross, P., et al.: A. M. A. Arch. Dermat. & Syph. 70:94, 1954

DOME CHEMICALS INC.

125 West End Avenue, New York 23, N. Y.
665 N. Robertson Blvd., Los Angeles 46, Cal.
2765 Bates Road, Montreal, Canada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than any other brand . . .
made to the high standards
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LIQUID MAKE-UP
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Incorporated in exclusive ACID MANTLE vehicle

Quick Soothing Topical Response

IN INFLAMMATORY DERMATOSES

Cort-Dome™

Acid Mantle-Hydrocortisone

CREME AND LOTION—pH 4.6

ABATES INFLAMMATION AND RESTORES NORMAL SKIN ACIDITY

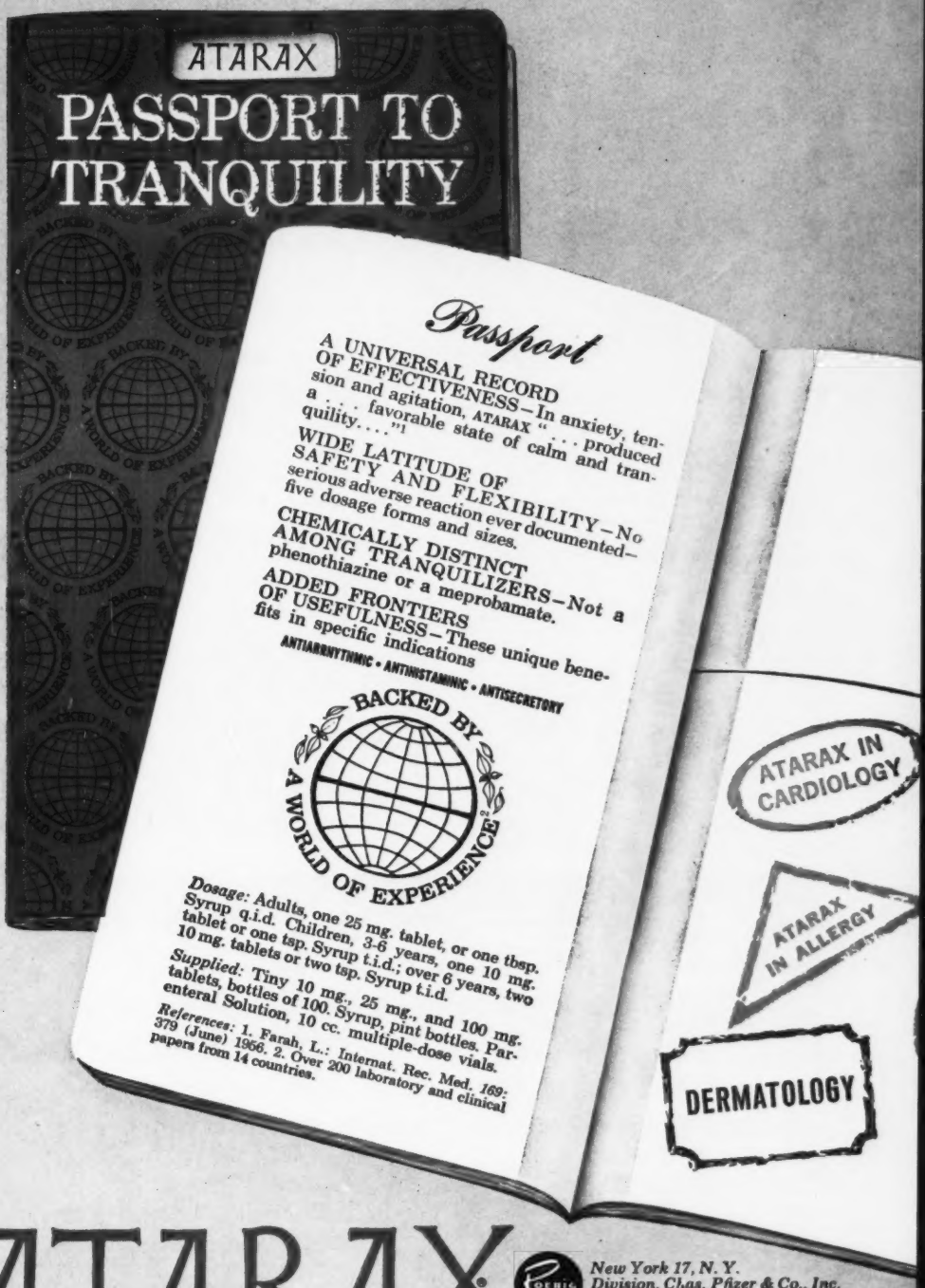
"Susceptibility to infection by bacteria is widely held to be due to the removal of the antibacterial 'acid mantle' of the skin and its displacement by an abnormal 'alkaline mantle'."—Fabricant, N. D.: A. M. A. Arch. Otolaryn. 65:11, 1957.

Supply—½%, 1% and 2% hydrocortisone in either Creme (½ oz., 1 oz., 2 oz., 4 oz. tubes and 1 lb. jars) or Lotion (½ oz., 1 oz., 2 oz., 4 oz. squeeze bottles and pt. bottles).

Samples and literature available on request.

Dome Chemicals Inc.

NEW YORK 23 • LOS ANGELES 46 • In Canada: 2765 Bates Road, Montreal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Passport
A UNIVERSAL RECORD OF EFFECTIVENESS—In anxiety, tension and agitation, ATARAX "... produced a ... favorable state of calm and tranquility...."[1]
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CHEMICALLY DISTINCT AMONG TRANQUILIZERS—Not a phenothiazine or a meprobamate.
ADDED FRONTIERS OF USEFULNESS—These unique benefits in specific indications
ANTIARRHYTHMIC • ANTIHISTAMINIC • ANTISECRETORY
BACKED BY A WORLD OF EXPERIENCE
Dosage: Adults, one 25 mg. tablet, or one tbsp. Syrup q.i.d. Children, 3-6 years, one 10 mg. tablet or one tsp. Syrup t.i.d.; over 6 years, two 10 mg. tablets or two tsp. Syrup t.i.d.
Supplied: Tiny 10 mg., 25 mg., and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.
References: 1. Farah, L.: Internat. Rec. Med. 169: 379 (June) 1956. 2. Over 200 laboratory and clinical papers from 14 countries.
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ATARAX®

(brand of hydroxyzine)

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Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

Never before in **ACNE!**

Graded long-term abrasion

The 3 grades of BRĀSIVOL permit a logical 3-step course of therapy:

1

Mild abrasion for 2-4 weeks with BRĀSIVOL FINE

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More vigorous abrasion for 2-4 weeks with BRĀSIVOL MEDIUM

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Maintenance treatment for as long as needed with BRĀSIVOL ROUGH

BRĀSIVOL introduces a new concept in the treatment of acne: prolonged superficial abrasion precisely adjustable to the patient's progress. Ten years of clinical studies in over a thousand acne patients have demonstrated the success of continuous abrasion in maintaining patency of the sebaceous ducts by gentle desquamation, thereby reducing comedones, papules, pustules and cysts, and minimizing post-acne scarring.

BRĀSIVOL contains non-silicon abrasives (fused synthetic aluminum oxide) of carefully selected particle sizes and in appropriate ratios, with hexachlorophene 1.0%, in a specially formulated base of soap and synthetic detergent.

1. Saperstein, R. B.: Presented at 107th annual meeting of American Medical Assn., San Francisco, Calif., June 23-27. 1958.

SUPPLIED:

Jars of Brāsivol Fine 5¾ oz.; Brāsivol Medium 6¼ oz.; Brāsivol Rough 7 oz.

Brāsivol™

GRADED ABRASIVE CLEANSER FOR ACNE

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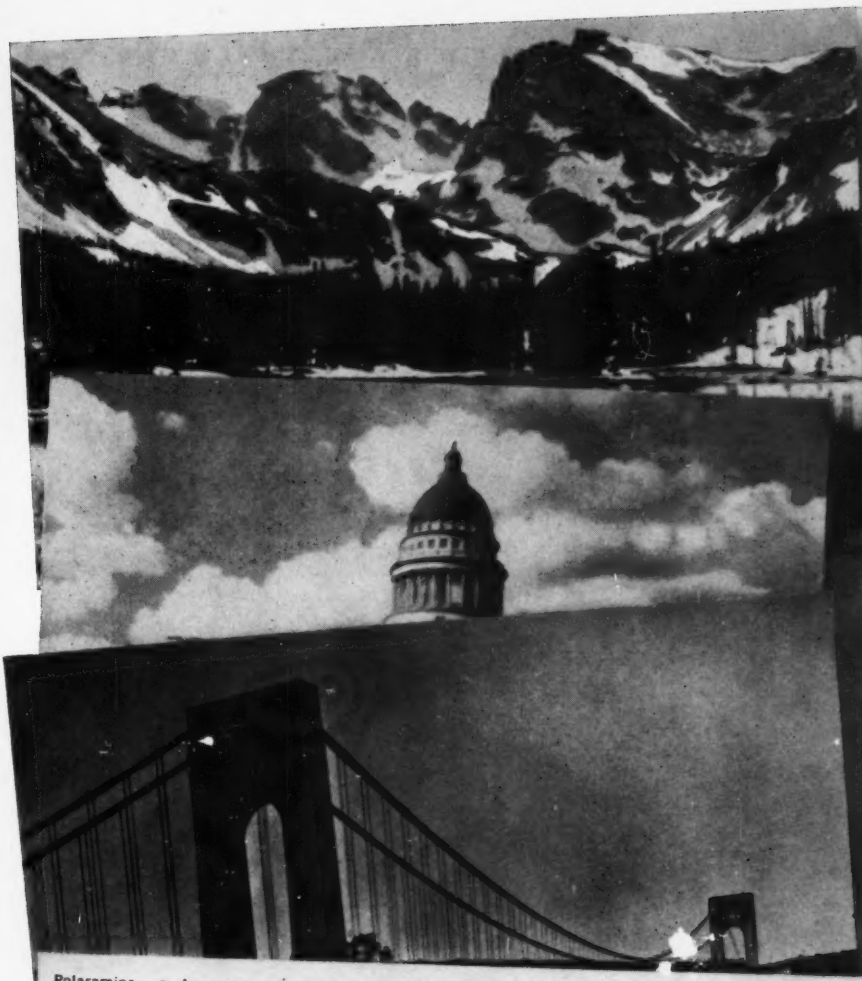

Polaramine—a close approach to a perfect antihistamine— offers four major benefits to provide your patients with unsurpassed freedom from seasonal and nonseasonal allergies.

Unsurpassed therapeutic response ... daylong or nightlong relief with a single 4 mg. Polaramine Repetab. Excellent protection at lower dosages than with other antihistamines. Unexcelled safety ... highest therapeutic index (3380) of almost all antihistamines. Notable freedom from side effects. 4 mg. Polaramine Repetabs in bottles of 100 and 1000.

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REPETABS,® Repeat Action Tablets.

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SYMBOL OF THE ONE-DOSE CONVENIENCE YOU WANT FOR YOUR PATIENT

PROMPT REPETAB® RELIEF • LASTS ALL DAY OR NIGHT •

Wherever the 4 winds blow... new 4 mg.

POLARAMINE REPETABS

for relief of allergic reactions and day-to-day maintenance

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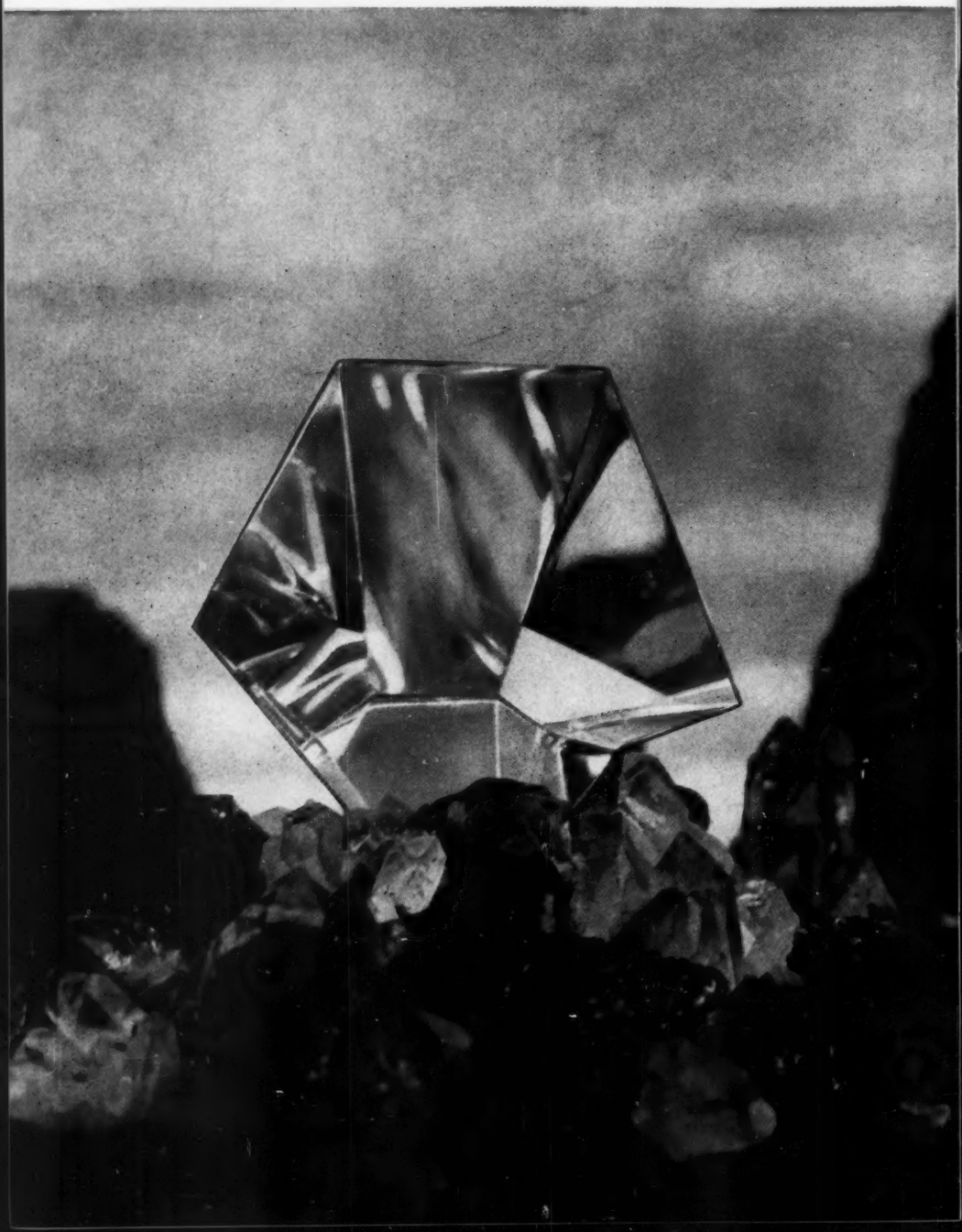
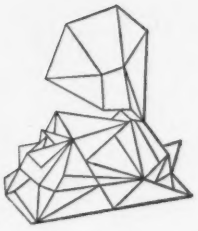
PURE ANTIHISTAMINE ACTION
A PHARMACOLOGIC FACT
BECAUSE DĪSOMER
SHEDS THE MOLECULAR DROSS



NEW...IN THE TREATMENT OF ALLERGIC DISORDERS



- *"...high therapeutic index"*[1]
- *unsurpassed clinical efficacy*
- *highly effective in exceptionally small doses*
- *side effects reduced to minimal level*

Disomer....a scientific contribution in the pharmacology of antihistamines!

Incorporating the newest knowledge of structure-function relationships, DISOMER comes close to the therapeutic ideal of pure antihistamine activity. DISOMER represents the *d*-isomer of racemic brompheniramine maleate. In shedding the *l*-isomer a high point in clinical effectiveness is achieved while side effects are reduced to a minimal level.

Therapeutic results have been noteworthy with 94.7% effectiveness reported.[2] Equally noteworthy is the low incidence of clinically significant adverse reactions. Indeed, the sole side effect reported was occasional, mild drowsiness in only 4.7% of patients.

With DISOMER your allergic patient remains your alert patient while enjoying unsurpassed freedom from allergic symptoms. Ready now for your prescription—DISOMER is available in a variety of dosage forms to fit your patients' individual requirements.

Availability:

DISOMER CHRONOTAB*	6 mg.
DISOMER CHRONOTAB*	4 mg.
DISOMER Tablets	2 mg.
DISOMER Syrup	2 mg. per 5 cc.

Usual dosage:

6 mg. CHRONOTAB	b.i.d.
4 mg. CHRONOTAB	t.i.d.
2 mg. Tablet	q.i.d.
Syrup 1 teaspoonful	q.i.d.

**Chronotab* is White's repeat-action tablet.*

References: (1) Gould, A. H. and Long, D. L.: Clinical Pharmacology and Therapeutic Use of Dexbrompheniramine Maleate (Disomer), a new Histamine Antagonist (submitted for publication). (2) Medical Department, White Laboratories, Inc.

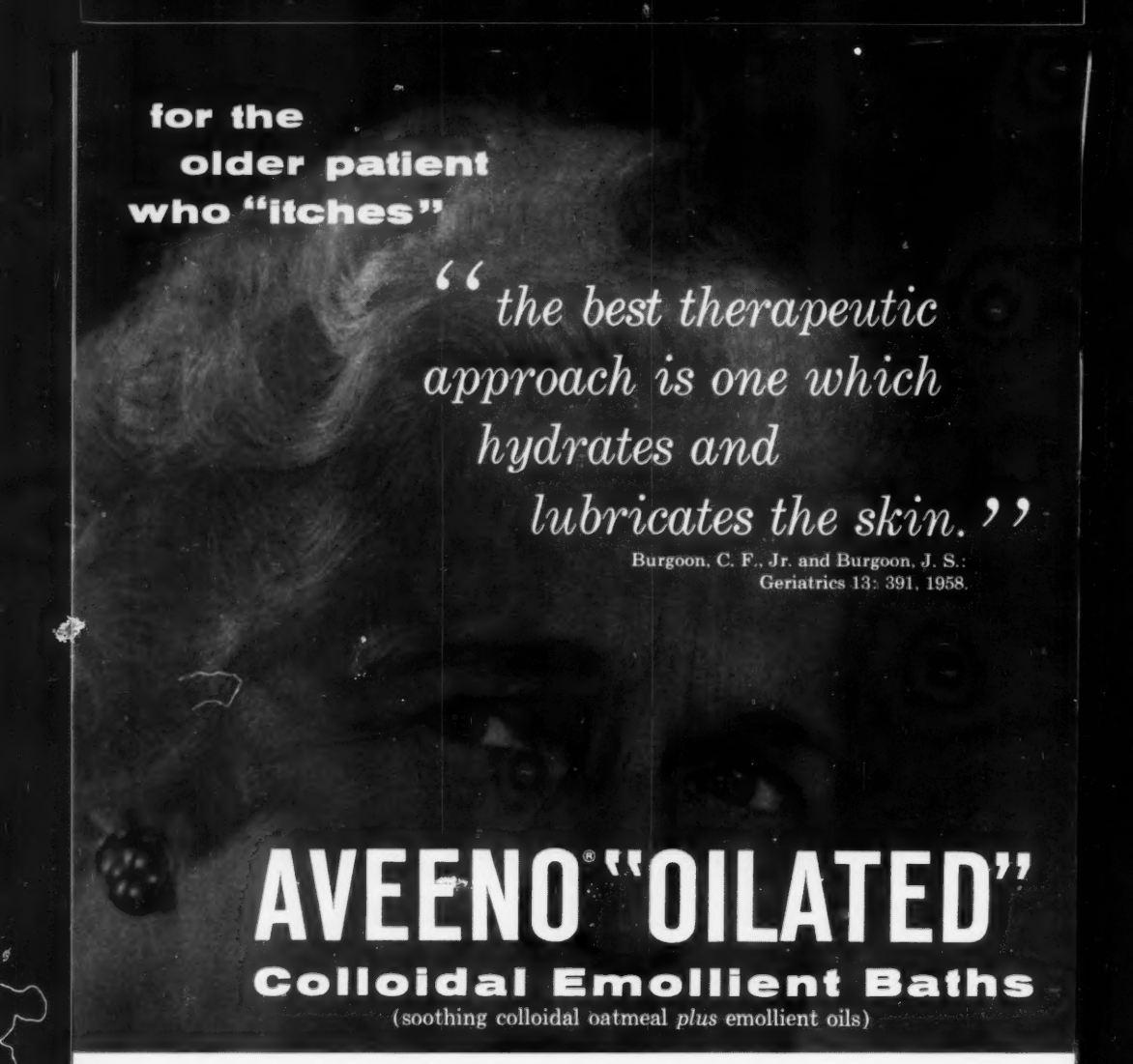
WHITE LABORATORIES, INC.
Kenilworth, New Jersey

White's

DISOMER

DEXBROMPHENIRAMINE MALEATE

...sheds the molecular dross


for the
older patient
who "itches"
"the best therapeutic approach is one which hydrates and lubricates the skin."
Burgoon, C. F., Jr. and Burgoon, J. S.:
Geriatrics 13: 391, 1958.

AVEENO® "OILATED"

Colloidal Emollient Baths

(soothing colloidal oatmeal *plus* emollient oils)

hydrate the skin Tepid colloidal baths supply water of hydration to the dry epithelium.

lubricate the skin Because Aveeno "Oilated" contains 35% emollient oils, the oil film left on the skin retards water loss from the stratum corneum . . . and, at the same time, provides *extra* soothing and antipruritic qualities due to colloidal oatmeal.[1-3]

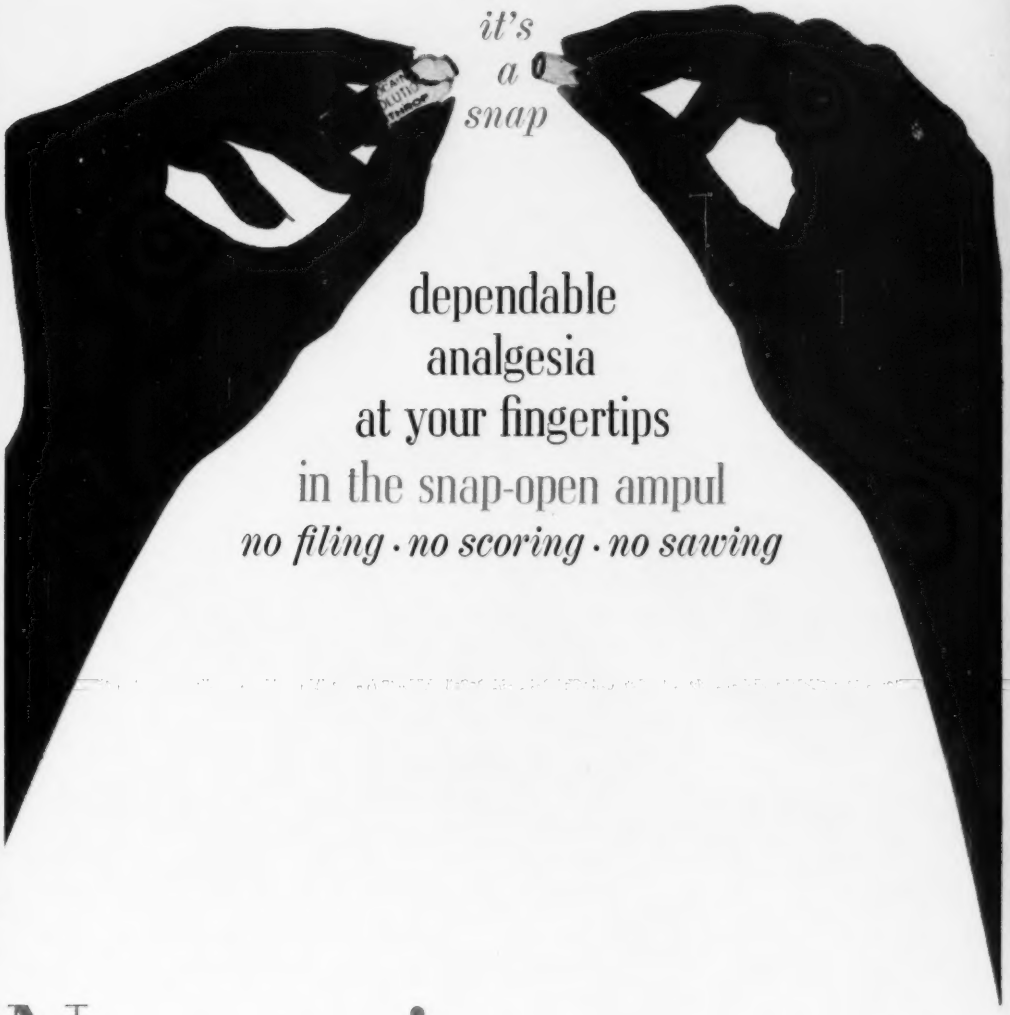
Indications: Dry skin, senile pruritus, bath itch, pruritus ani and vulvae, cold weather pruritus.

AVEENO® "OILATED" is available in 10 oz. cans.

Active Ingredients: Colloidal oatmeal concentrate, liquid petrolatum and hypo-allergenic fraction of lanolin.

References: 1. Sulzberger, M. B. and Wolf, J. Dermatology. Chicago, Year Book Publications, Inc., 1952, p. 42. 2. Grais, M. L.: A.M.A. Arch. Dermat. 58: 402, 1953. 3. Kierland, R. R. and Ede, M.: A.M.A. Arch. Dermat. 63: 602, 1951.

AVEENO CORPORATION 250 West 57th Street, New York *Pioneers in Ethically-Promoted Colloid Baths*



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PIONEER BRAND OF PROCAINE HYDROCHLORIDE

a local anesthetic with universal acceptance

Novocain 1%, 2%, 10%, 20% Solutions with or without vasoconstrictors.
Also available: Multiple Dose Vials with dual purpose caps for withdrawal by needle or pouring.

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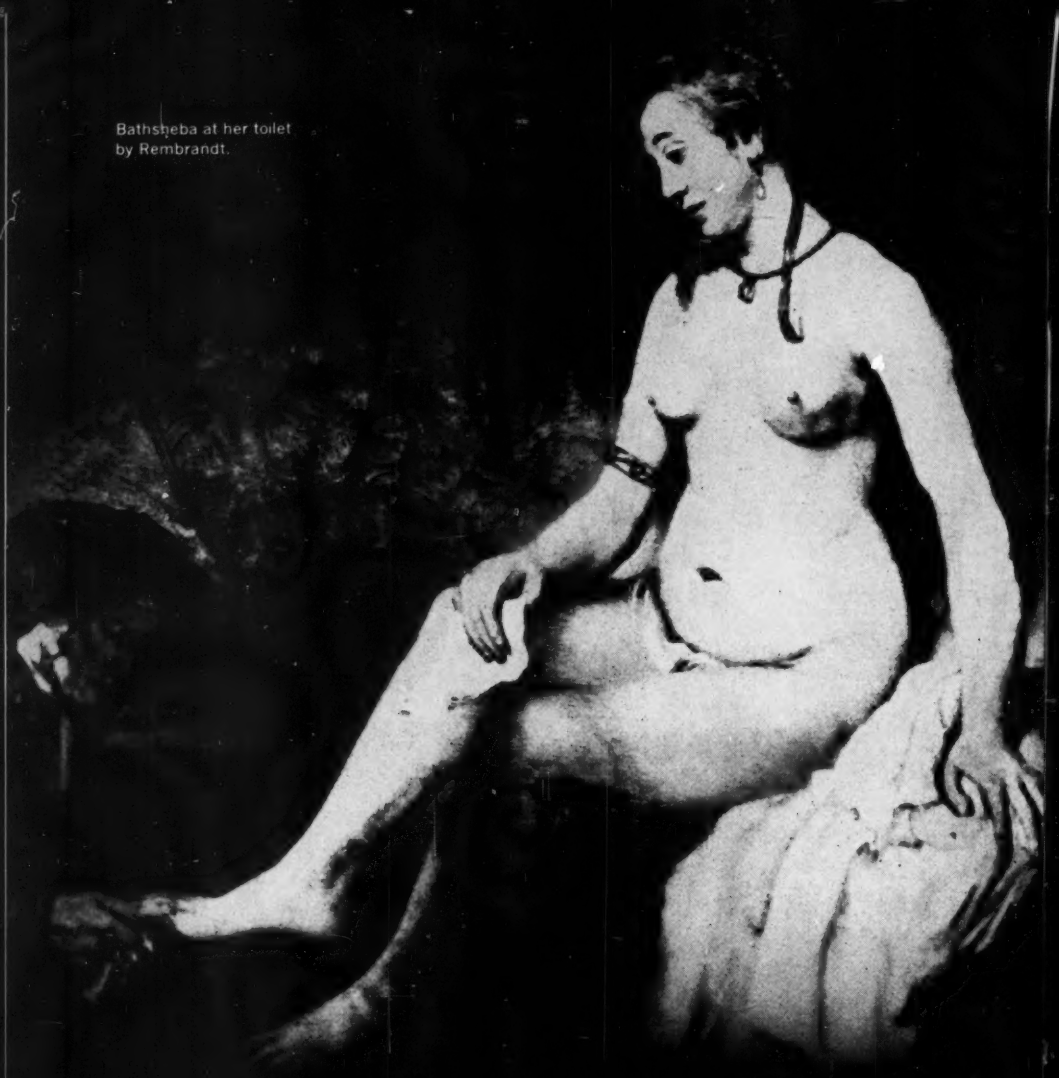

Bathsheba at her toilet
by Rembrandt.

a pleasant way to treat dry, itchy skin

Alpha-Keri™

water-dispersible, antipruritic oil for the bath or shower

Alpha-KERI makes dry skin feel soft and smooth immediately. It effectively deposits a uniform, partially-occlusive oil film over the entire skin area. Alpha-KERI lubricates the skin, relieves itching and restores the protective action of natural skin oils lost by the action of water, weather and detergents. It moisturizes the skin and also helps to retain moisture by retarding evaporation of water. Alpha-KERI contains: Kerohydric™, brand of dewaxed, oil-soluble, keratin-moisturizing fraction of lanolin, mineral oil, and a special nonionic emulsifier which provides the right amount of water dispersibility for optimum coverage of the skin with emollient oils. Alpha-KERI oil may be used in the bath, in the shower, for sponge bathing and for infant baths. It can also be used for skin cleansing where soap is contraindicated. Alpha-KERI oil is tinted an attractive green color and pleasantly scented. Bottles of 8 fl. oz. *Write for samples and literature.*

Westwood Pharmaceuticals, Buffalo 13, New York

NEW

for "a particular problem"[1]

PSORIASIS OF THE SCALP

a specific... superior... solution:

SARCOPHEN™

clinically effective

cosmetically adequate

Sarcophen produced satisfactory improvement in 97% of 138 patients with psoriasis of the scalp.[2]

The unique Sarcophen formula softens scales without need for harsh irritants. It is notably stable and homogeneous—entirely different from the usual phenol-mineral oil mixtures.

Sarcophen is a clean liquid that won't stain, won't give hair a greasy look . . . indeed, Sarcophen makes a fine hair dressing in psoriasis.

Contains phenol 1.2% in distilled acetylated monoglycerides with isopropyl alcohol, specially formulated for scalp application. Bottles of 4 fluid ounces.

1. Pillsbury, D. M., Shelley, W. B., and Kligman, A. M.: Dermatology. Philadelphia, Saunders, 1956. P. 733.
2. Goldman, L., and Preston, R. H.: A.M.A. Arch. Derm., 80:222 (Aug.) 1959.

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Atopic dermatitis before treatment

NOW...
to relieve inflammation fast

- mg. for mg. the most active steroid topically—up to 40 times the potency of hydrocortisone
- optimal not minimal steroid concentration for peak effectiveness . . . maximal contact at the site of the lesion
- stops the itch-scratch cycle to aid inflammation relief and maintain patient comfort day and night
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- no irritating steroid particles, no sting, stain, smell, stickiness

After treatment

ACTUAL CLINICAL PHOTOGRAPHS

TOPICAL CREAM

NeoDecadron*

DEXAMETHASONE 21-PHOSPHATE—NEOMYCIN SULFATE

INDICATIONS: Allergic or inflammatory dermatoses, with or without pruritus; sunburn; insect bites; otitis externa (only if the drum is intact).

CAUTION: Steroids should not be used in the presence of tuberculosis of the skin.

DOSAGE: A small quantity of NeoDECADRON Topical Cream (0.1%) is applied to the affected area 2-3 times daily.

Additional information is available to physicians on request.

*NeoDECADRON and DECADRON are trademarks of Merck & Co., Inc.

Product	Steroid Concentration	ACTIVE INGREDIENTS: Dexamethasone 21-Phosphate (as disodium salt)	ACTIVE INGREDIENTS: Neomycin Sulfate	Supplied
NeoDECADRON Topical Cream	0.1%	1 mg./Gm.	5 mg./Gm. (equivalent to 3.5 mg. neomycin base)	5 Gm. (⅙ oz.) tube 15 Gm. (½ oz.) tube
DECADRON® Phosphate	0.1%	1 mg./Gm.	—	5 Gm. (⅙ oz.) tube 15 Gm. (½ oz.) tube

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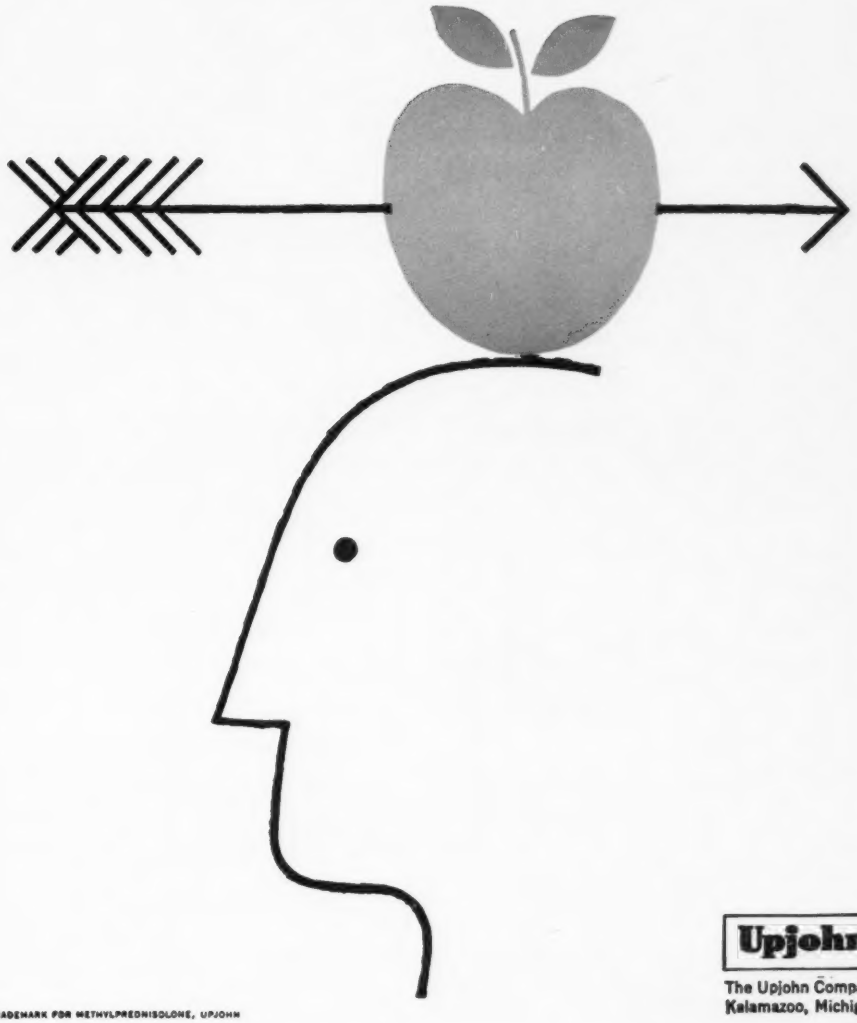
Mildness is only part of the Ivory Soap story

Not just mildness but uniform quality makes Ivory a soap dermatologists can trust. Each bar is pure white and pure scented. Each is gentle enough for even delicate baby skin. Only the most careful control and continuous testing make this consistent purity and gentleness possible. When a pure, bland soap is indicated for your adult, adolescent, or baby patients—you can recommend Procter & Gamble's Ivory Soap.

99 44/100% pure® . . . it floats

Medrol*

hits the disease but spares the patient



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The Upjohn Company
Kalamazoo, Michigan

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to make grateful psoriasis patients...

coal tar therapy with cosmetic consideration

WRIGHT'S®

Liquor Carbonis Detergens

This pleasant English coal tar solution is a time-proven dermatologic standard of reliability on two continents. In elegance, it is superior to the usual crude coal tar preparations.

Whether you prescribe WRIGHT'S L.C.D. for application in its undiluted form or by incorporation in ointments and lotions, your patient will be pleased with its ease of applicability and removal, the absence of greasy staining.

WRIGHT'S L.C.D. is especially useful for tar baths in generalized dermatoses.

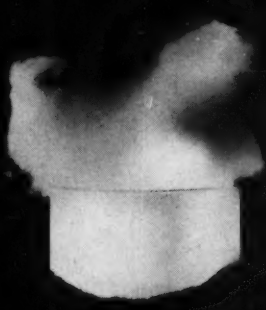
Indicated in chronic psoriasis, atopic dermatitis, chronic eczematous disorders and many other dermatoses.

WRIGHT'S LIQUOR CARBONIS DETERGENS is available in 3-oz., 8-oz. and 16-oz. bottles; also in the new 2-oz. aerosol topical spray, the modern method of application where only the medication touches the lesion.

A complimentary trade-size dispenser of the aerosol is yours when requested on your letterhead.

FOUGERA Hicksville, Long Island, New York

"...an effective and an aesthetic form of therapy for patients with



seborrheic dermatitis of the scalp and pyodermas of the skin"[1]

unsurpassed broad-range germicide plus detergent action

BETADINE™ SHAMPOO

(active ingredient 1% ... iodine)

checks infection • scaling • flaking • itching

BETADINE SHAMPOO successfully treats seborrheic conditions characterized by erythema, scaling and exfoliation, excessive dandruff, and pruritus. BETADINE SHAMPOO, for daily over-all cleaning, is an effective prophylactic agent in preventing acute inflammatory skin conditions caused by pyogenic organisms.

BETADINE SHAMPOO is nonstaining and virtually nonirritating.

supplied: BETADINE SHAMPOO in 4-oz. plastic squeeze bottle, complete with directions.

1. Frank, L.: Research Report 1:35

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Come visit our Exhibit at the American Academy of Dermatology and Syphilology Meeting in Chicago, December 5-10. See Two New Applicators — the Helsper Linear Applicator to remove keloids and linear scars, the Cervical Lesion Applicator to treat endocervicitis. Booth 79.

MINIMAL PAIN
MINIMAL SCARRING
MINIMAL COST

In the treatment of
PLANTAR WARTS
and other skin lesions

the **KIDDE** DRY ICE APPARATUS

The excellent cosmetic results to be obtained with cryotherapy in obliterating verrucae, nevi, angiomas, cystic acne, etc., are well recognized.* But dry ice is cumbersome to obtain and handle.

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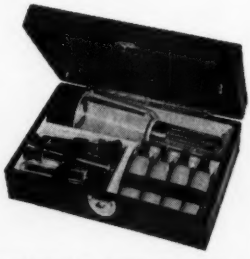

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*J.A.M.A. 118:296, 1942.

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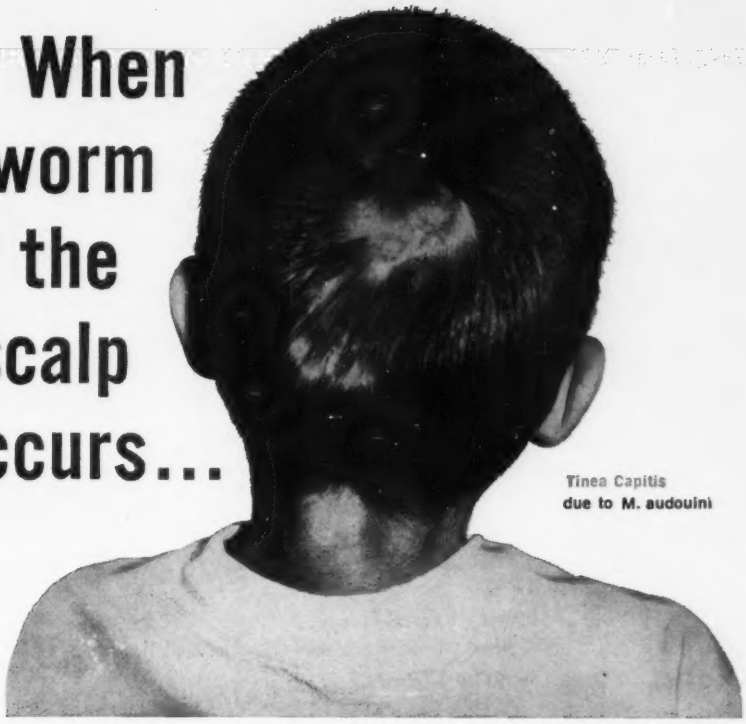

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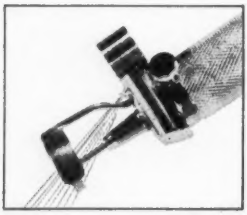

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*A.M.A. Archives of Dermatology, April 1955
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A.M.A. ARCHIVES OF
DERMATOLOGY

The Interrelation of Some Cutaneous and Ocular Diseases

Particular Discussion of Blepharochalasis, Sarcoid, Erythema Multiforme, Ocular Pemphigus, Lupus Erythematosus, Melanosis of Conjunctiva, Length of Cilia

JOSEPH V. KLAUDER, M.D., Philadelphia

The dermatologic clinic at Wills Eye Hospital affords opportunity of observing cutaneous diseases confined to the lids or conjunctiva, or with concomitant ocular manifestation.

Ocular examination may be of diagnostic aid to the dermatologist. A notable example is leprosy, a disease that has according to deBarros [1] a higher percentage of ocular involvement than any other systemic disease. Of its diverse ocular lesions, one constitutes one of the earliest signs of leprosy. Reference is made to beaded corneal nerve as seen by slit-lamp, microscopy.[2] Second in diagnostic importance are distinctive capillary changes seen in the capillary bed at the limbus. Elliot [2] compared these changes to that of a glomerulus or capillary tufts.*

Submitted for publication April 14, 1959.

From the Department of Dermatology, Wills Eye Hospital (Dr. Joseph V. Klauder, Chairman), and Department of Dermatology, Graduate School of Medicine, University of Pennsylvania (Dr. Herman Beerman, Chairman).

* There is considerable anatomical similarity between the terminal vascular network of the capillary bed in the limbus of the cornea and the capillary bed of the skin. It would therefore be pertinent in study of capillary changes in the skin to include the limbus of the cornea. This would also apply in study of capillary changes in acute lupus erythematosus.

Of considerable aid in diagnosis of congenital syphilis in later years is the demonstration of old blood vessels of interstitial keratitis by corneal examination and slit-lamp microscopy. Such blood vessels, characteristically presented, remain indefinitely, and may constitute the only evidence of congenital syphilis.[3]

Perivasculitis in a child, or young adult, is of import in diagnosis of congenital syphilis.[4]

The presence of an active or inactive uveitis is of help in diagnosing sarcoidosis; likewise, angioid streaks of the retina in diagnosis of pseudoxanthoma elasticum. Diagnosis of recurrent genital and oral aphthae may be facilitated by ocular examination to demonstrate an eye lesion that is part of Bechet's syndrome.[5] Iridocyclitis is preceded by recurring attacks of conjunctivitis that increases in severity. Diagnosis is frequently missed since the patient receives only a specialized examination, by either a dermatologist, ophthalmologist, or gynecologist. Each of these specialists should have a high index of suspicion of this disease.

Some other cutaneous diseases in which ocular examination is indicated would include the following: characteristic lenticular changes of atopic dermatitis,[6] retinopathy

of acute lupus erythematosus, and arterial changes in the chronic form of the disease; retinal tumors, retinal detachment, and choroiditis associated with tuberous sclerosis,[7] and the diverse ocular lesion of xeroderma pigmentosum.[8] Retrolental fibroplasia should be considered in infants with cutaneous angiomas; as well as optic neuritis and lenticular changes caused by pellagra, and night blindness, keratomalacia, and xerophthalmia associated with vitamin A deficiency.

Fundal changes are common in all types of leukemia. These comprise engorged and tortuous retinal vessels, hemorrhages; later a typical ophthalmoscopic picture is presented—leukemic retinopathy. In polycythemia ocular complications are not uncommon; they comprise hyperemia of the bulbar conjunctiva, engorgement and tortuosity of the retinal veins, a cyanotic fundus, retinal hemorrhages, edema of the disk, and thrombosis of the central retinal vein.

In sickle-cell anemia, changes in the optic fundus comprise tortuosity of the blood vessels, retinal edema, and at times pallor of the disk. Hanno[9] observed three patients at Wills Hospital with sickle-cell disease who had spontaneous vitreous hemorrhages. It was bilateral in two patients, one of whom had bilateral retinitis proliferans.

An intensive conjunctivitis accompanies the oculoglandular type of tularemia. Blindness may result from this disease. This is by reason of perforation of a corneal ulcer, and also the occurrence of bilateral optic neuritis.

Minor ocular symptoms are commonly associated with rosacea—blepharitis and blepharoconjunctivitis. Less common ocular concomitants comprise nodular infiltrations of the conjunctiva, cornea, and rarely the episcleral tissues. These are regarded as specific lesions of the disease.

Rosacea keratitis, especially in its severe form and its tendency to recur, is the most serious and may lead to considerable impairment of vision. Thygeson[10] gives 5% as the incidence of severe keratitis. The first appearance of keratitis is a marginal vascular infiltration; the next stage is the development of subepithelial infiltrates; they slowly progress and are heavily vascularized. Eventually, the epithelium over them becomes eroded with the formation of an ulcer. These corneal changes are pathognomonic of rosacea keratitis.

Of viral diseases the following would be included:

Diverse ocular lesions have been attributed to the virus of lymphogranuloma venereum. These comprise edema of the lids, conjunctivitis, episcleritis, iritis, retinal hemorrhages, corneal ulcer, peripapillary edema, and tortuous blood vessels. Lymphogranuloma was suspected as a cause of keratoconjunctivitis of patients reported by Macnie.[11] He attributes the virus as one of the causes of the oculoglandular syndrome of Parinaud. Macnie experimentally produced uveitis and keratoconjunctivitis in guinea pigs by injection of mouse brain emulsion of the virus.

Some are of the opinion that the ocular symptomatology of lymphogranuloma venereum has not as yet become established. Benedek and Olkon[12] pertinently write: "It is extremely difficult to evaluate the connection of disease of the eye with lymphogranuloma venereum."

One ocular lesion that is, perhaps, more generally accepted as caused by lymphogranuloma venereum is a distinctive form of keratitis. This affects the entire depth of the cornea with superficial blood vessels, resembling an epaulette. The lesions extend onto the cornea at the upper limbus; the lower border of the vascular area is sharply marginated. Early diagnosis and treatment is necessary to prevent permanent corneal damage.

Herpes simplex[13] may occur on the lid and lid margins, or, as a primary ocular herpes causing severe conjunctivitis with preauricular lymphadenopathy resembling the changes in epidemic keratoconjunctivitis. Corneal involvement is variably pre-

sented—dendritic ulcer, stellate keratitis, diskiform keratitis. These may occur with or without herpetic lesions elsewhere. Herpetic iritis is a frequent accompaniment.

If lesions of herpes zoster ophthalmicus extend to the tip of the nose indicating involvement of the lachrimal and nasociliary branches, as originally pointed out by Hutchinson, involvement of the cornea (herpetic keratitis) is to be expected. An exception is an anomalous distribution of these nerves. Conjunctivitis, secondary to herpetic lesions on the conjunctiva, rarely occurs.

Conjunctival and corneal involvement occurs in varicella, variola, and in cowpox (milkers' nodules). In addition to conjunctivitis, optic neuritis may occur in infectious mononucleosis.

Molluscum contagiosum may occur on the lid and lid margins. In this event, conjunctivitis is a frequent occurrence; it is of the follicular type and often accompanied by keratitis. In prevention of ocular involvement, prompt removal of lesions on the lid is desirable. Such removal facilitates disappearance of ocular involvement.

Considering the frequency of vaccination, ocular involvement is rare. It occurred in 19 persons of 850,000 persons vaccinated, as reported by Sedan, Aurgaud, and Guillot.[14] Of ocular involvement, vaccinia of the lid is most frequent. This occurs as autoinoculation (as shown in Figure 1), or, as heteroinoculation, in which the virus is transferred from one person to another. According to Moffatt [15] this form pursues a more severe course.

In the heteroinoculation in which diagnosis is less apparent, it should be suspected in a slowly developing vesicular, pustular lesions on the eyelid. True vaccinia pustule on the conjunctiva [15] or cornea is rare and is more serious.

On the eyelid there are an essociated blepharitis and conjunctivitis; symblephara may result, and a vaccinial disciform keratitis. Moffatt [15] gives the incidence of corneal involvement from 10 to 30%. Visual impairment often results from such complication.

When vaccination is performed, instruction should be given in the direction of preventing auto- and heteroinoculation. Some writers recommend vaccination on the back of the arm, a site less likely to be touched by the fingers.

Cutaneous Lesions

Dermatologic Examination of Aid in Ophthalmologic Diagnosis.—On the other hand, examination of the skin and mucous surfaces may be of aid in ophthalmologic diagnosis. Establishing the diagnosis of von Recklinghausen's disease may reasonably explain diverse ocular manifestations of intracranial lesions of this disease. A pertinent illustration was the presence of a few *café-au-lait* spots on a young man who had atrophy of the optic nerve of obscure origin. Cutaneous lesions of sarcoidosis support ocular diagnosis of this disease. Cutaneous and mucosal lesions of pemphigus likewise support diagnosis of ocular pemphigus. The presence of symblepharon with irritative symptoms may reasonably be explained from a history suggestive of Steven-Johnson syndrome. The aforegoing discussion of other cutaneous diseases with concomitant ocular manifestation is pertinent in ophthalmologic diagnosis.

Cutaneous Diseases and Blindness.—I may mention here some cutaneous diseases in which the dermatologist is vitally concerned in prevention of blindness, or impaired visual acuity—sarcoidosis, severe forms of erythema multiforme (Steven-Johnson syndrome), and ocular pemphigus. Each exerts its relentless toll of vision. Leprosy and syphilis may be included in this category. As a result, however, of modern therapeusis of leprosy and the diminishing incidence of syphilis, these diseases are now of less import in the cause of blindness. Other rare cutaneous diseases could be included; for example, the devastating ocular lesions of xeroderma pigmentosa.

Cases from Wills Hospital

Blepharochalasis.—This is defined in the dictionary as relaxation of the skin of the eyelid due to atrophy of the intercellular tissue. Clinically, in its broadest meaning the term designates redundant, loose, flabby lids, usually affecting both upper and lower lids, and usually bilateral. It is more simply termed baggy lids. There are diverse causes of baggy lids.

From my observation, and for purposes of discussion, blepharochalasis is classified in the following four types: (1) Fuchs type, (2) senile, (3) hereditary, and (4) undetermined. Types 3 and 4 are not clearly defined in text books or in the literature.

Blepharochalasis as usually discussed in text books is confined to the restricted meaning of the term, to indicate what is here designated as the Fuchs Type.[16] Although this type was first described by Beers in 1807, it became well known through a comprehensive report by Fuchs in 1896, who proposed the term blepharochalasis.

This type is characterized by recurring attacks of edema, usually of the upper lids, resulting in a laxity of the lid tissue with dilation of the vessels, with thinning, wrinkling, discoloration of the skin, with finally a prolapse of orbital fat and the tear glands, and dropping of the lids. Its occurrence at middle age and older is infrequent, as is involvement of the lower lids.

At onset edema is transitory, is usually of short duration, and resembles angioneurotic edema. Later, the swelling becomes constant with bagginess of the skin that hangs loosely over the lid margins. At this stage the subcutaneous tissue diminishes in bulk and in elasticity.

There are a number of reports[17,18] of the association of blepharochalasis with double upper lid.

Histologic examination, as reported by different writers, shows an increased number of blood vessels, endothelial proliferation and infiltration of round cells. Some writers report absence or atrophy of the elastic fibers, others have observed no decrease. Histologic study of the aforementioned types of blepharochalasis is indicated.

The hereditary aspect of the Fuchs type of blepharochalasis is discussed by Panneton,[19] who observed 51 cases in three generations, and by Cockayne.[20] The latter author classifies the disease in his group of abiotrophies of elastic tissue; in this group he includes pseudoxanthoma elasticum, to which, he believes, the disease is closely allied.

Fig. 1.—Vaccinia of lid.

The patient shown in Figure 2 is probably an instance of the disease. The patient's age and involvement of the lower lids, however, oppose this diagnosis. In this patient the process was not regarded as angioneurotic edema or elephantiasis nostras. Melkerson-Rosenthal syndrome was considered, although there were no other symptoms of this syndrome.

The senile type of blepharochalasis is the most frequent type and is well known (Fig. 3). I believe a considerable percentage of all persons past middle age, and older, have some degree of blepharochalasis. It may, or may not, be associated with other objective signs, especially the skin, of the aging process. It is doubtless caused by loss of elasticity that accompanies the latter process. For purposes of classification of blepharochalasis it is difficult to state the age at which the designation "senile" is not appropriate. In consideration of this, I designated an "undetermined" group. An instance of this is shown in Figure 4. Blepharochalasis of this patient appeared at the age of 42 and was unaccompanied by other objective signs of advancing age, unless xanthelasma, which the patient presented (shown in Figure 4) is regarded as such. Patients with blepharochalasis in this group do not fit in the other three categories.

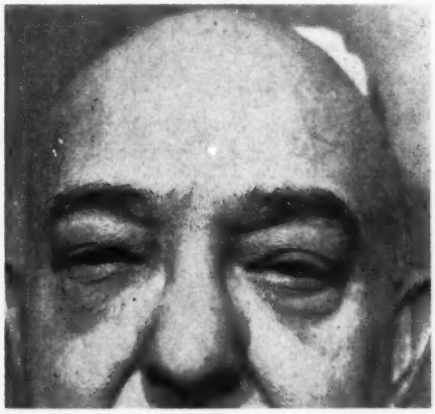

Fig. 2.—Blepharochalasis (probably Fuchs type). The patient is age 52. Its onset was at age 48 with recurrent attacks of swelling of both lids of the left eye; the degree of swelling varied and at times was absent for several months. At age 51 both eyelids of right eye became affected and pursued a similar course.

Fig. 3.—Blepharochalasis (senile type). A man age 64 for 10 years has had redundant, flabby, loose skin of upper and lower lids of both eyes. No history of recurrent attacks of swelling. The process is more pronounced now than at onset.

At times blepharochalasis is seen in patients who give a history of a familial

Fig. 4.—Blepharochalasis (undetermined type). Swelling of the lids of both eyes appeared five years prior at age 42 and has remained constant. There was no familial history of blepharochalasis and no signs that characterize ageing process, with possible exception of xanthelasma.

Fig. 5.—Blepharochalasis (hereditary type). Involvement of the lids of this 56-year-old patient has remained unchanged since onset in his early thirties. There is history of similar involvement of his father, one paternal uncle, and patient's son (age 30). His three daughters (the eldest is 25) are unaffected.

incidence. This is represented in the patient shown in Figure 5. I know of no reported study of a large series of such patients.

Blepharochalasis is generally attributed to diminished muscle tone, vasomotor states, atrophic changes in the epidermis or subcutaneous tissues, familial traits, or herniation of periocular adipose tissue through an impaired tarso-orbital fascia.

Other causes of the baggy eyelids are: edema, retrobulbar tumors, hyperthyroidism, myxedema, chronic nephritis, elephantiasis nostras, von Recklinghausen's disease, and dermatomegaly (cutis laxa).

Sarcoid (Besnier-Boeck-Schaumann Disease).—The patient shown in Figure 6 was a male Negro, aged 21. The lesions shown in the photograph affected the right eye and were of 14 months' duration. The rounded lesion on the external portion of the lower lid was firm, circumscribed, and felt like rubber. On the internal portion of the upper lid there were a few split-pea-sized lesions and an elevated one that extended over the canthus. The lens of the left eye was opaque. There was an old iritis with synechia-pthisis bulbi. Visual acuity was nil. These changes were attributed to sarcoidosis.

Roentgenograms of the hands showed characteristic changes of sarcoidosis in the distal ends of the phalanges; that of the lungs showed no abnormalities.

In Wegner and Wurm's [21] study of 172 cases of sarcoid, 130 were females and 42 males. The age range was 10 to 70, and the predominating range was 20 to 35. Of the 172 patients, 161 had iridocylitis; in 36 there was choroiditis. Nodes on the iris were present in 77 patients. In only 25

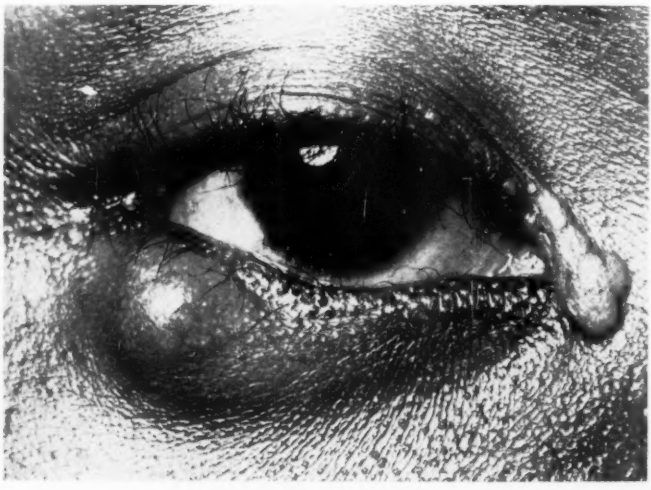

Fig. 6.—Sarcoid of lids of right eye. Sarcoidosis of left eye resulted in pthsis bulb.

patients, ocular involvement was unilateral. The interval between first and second eye involvement varied from 8 days to 22 years. In about two-thirds of the patients, on account of ocular involvement, sarcoid was first diagnosed, and in 69 patients iritis was the first symptom of the disease. In 30% of their patients the lungs remained uninvolved. Severe forms of pulmonary involvement were without symptoms of such involvement.

Erythema Multiforme.—The devastating result of ocular involvement in severe forms of this disease (generally known in this country as Steven-Johnson syndrome) is shown in Figure 7.

For one year prior to admission, this patient complained of painful and irritable eyes that followed an acute illness. The history was typical of Steven-Johnson syndrome. Sudden onset of a generalized, vesicular, bullous eruption, high fever, and involvement of both eyes and mouth. She was hospitalized for five weeks.

On admission to Wills Hospital, visual acuity of the right eye was hand movements, of the left eye counting fingers at 1 ft. The cul-de-sacs of both eyes were partially obliterated, with obliteration of the superior cul-de-sac of the right eye. The conjunctivas of both eyes were thickened, scarred, and dry; there was an absence of tears. In both eyes there were symblepharа extending from upper and lower lids to the globe. Both upper and lower puncta of the right eye were involved in conjunctival scar tissue. Both corneas were dry, there was marked scarring, and they were semi-opaque throughout with extensive vascularization. The lids were thickened and adherent in large part to the globe. Figure 7 is that of the left eye.

Fig. 7.—Sequela of ocular involvement in severe form of erythema multiforme. Showing partial obliteration of cul-de-sac, scarred conjunctiva, symblephara, semi-opaque cornea. The other eye was similarly affected; the patient was industrially blind.

In a prior publication [22] I stressed the frequent involvement of the eyes in erythema multiforme. In the milder forms of the disease ocular involvement is presented as catarrhal conjunctivitis that disappears without sequela. In the more acute form, it appears as purulent conjunctivitis and, in the severe form, as pseudomembranous conjunctivitis. The latter is the most serious form; it may progress to corneal perforation and endophthalmitis; as a sequela there are varying degrees of permanent ocular damage (Fig. 7). Such sequelae are not infrequently seen among the Wills Hospital clientele.

In prevention of ocular damage, early diagnosis and treatment of severe erythema multiforme and early ophthalmologic consultation are most desirable.

Ocular Pemphigus (Pemphigus of Mucous Membrane).—The patient shown in Figure 8 is age 80. She was first seen at

Fig. 8.—Ocular pemphigus in a woman aged 80. Its onset was 34 years prior; blindness ensued 20 years later. The patient never had cutaneous lesions of the disease or involvement of other mucous surfaces.

Wills Hospital in 1928. The ocular process affected both eyes for about three years. There occurred partial obliteration of the cul-de-sacs, symblephara, haziness of the corneas with some opacities. Visual acuity of the right eye was 3/60, of the left eye 1/60. As an illustration of slow progression of this disease to blindness, visual acuity in 1938 of the right eye was 1/60, of the left eye, light perception; in 1942 that of the right eye was 1/2/60, left eye nil. The right eye became blind about 1945. The patient never had cutaneous involvement or involvement of other mucous surfaces.

Figure 8 shows considerably narrowed, palpebral fissures, adherence of the lids to the globe, an opaque cornea, and obliteration of the cul-de-sac. The right eye was similarly involved.

In a report by Klauder and Cowan[23] terminology of ocular pemphigus was discussed. The disease, as reported by different writers, was divided into three groups: (1) This embraced severe forms of erythema multiforme, variously termed by different writers; (2) a manifestation of pemphigus of the mucous membranes; (3) conjunctival involvement accompanying a number of skin diseases, most of which exhibit blebs.

Pemphigus of the conjunctiva (Group 2) is usually a disease of later life. Although there are published reports of its occurrence in early adult life, Klauder and Cowan stated the opinion that many of such cases should be assigned to their Group 1 or 3.

Pemphigus of the conjunctiva may be confined to the eye (Fig. 8). Ocular involvement may precede, accompany, or follow involvement of one or more of the mucous membranes and/or cutaneous lesions (bullae, blebs). In the latter event, the lesions are transitory and usually localized. A generalized bullous eruption is rare.

The earliest symptom is a simple catarrhal conjunctivitis that disappears and reappears for years. Later, there is evidence of shriveling of the conjunctiva, together with symblephara. These symptoms slowly progress over a period of years. Another course of the disease is the initial appearance of the latter in absence of irritative symptoms.

The disease may begin in both eyes. If unilateral, the second eye invariably becomes involved in an interval of less than two years.

In the absence of shriveling conjunctiva and symblephara the diagnosis cannot be made with certitude. If, however, lesions of the disease are present on the skin and mucous membranes with only catarrhal conjunctivitis, diagnosis of ocular involvement is reasonable.

The slow progress of the disease is to be stressed. I know of no effective treatment. If the patient lives sufficiently long, I believe blindness is inevitable (to wit, the patient in Fig. 8). Since the disease is one of later life, expectancy of life may be shorter than expectancy of blindness.

Chancre of Lid.—This is shown in Figure 9. The patient was a woman, aged 45. The edema involved all of the lower lid. The latter suggested anthrax; dark field examination, however, showed Treponema pallidum. The patient conducted a boarding house and handled soiled linen of male

Fig. 9.—Showing chancre of lid as discussed in the text.

Fig. 10.—Showing malignant melanoma as discussed in the text.

Fig. 11.—Chronic lupus erythematosus of upper and lower lid. There were no lesions of the disease elsewhere.

boarders. This was the only clue of source of infection.

Malignant Melanoma of Lid.—This is shown in Figure 10. It first appeared in this 50-year-old patient as a small "pimple" two years prior to admission. It increased in size, and became black one month before. The lesion was 3×1 cm., was elevated 1 cm. and was deeply pigmented; the surface was dry and crusted. It chiefly involved the upper lid margin. The palpebral conjunctiva of the upper lid was pigmented, extending into the formix and only slightly toward the midline. The bulb was uninvolved. The cornea was clear. The preauricular and cervical nodes were enlarged. Roentgenograms of chest and spine showed no evidence of metastasis. Histologic examination of the entire excised lesion was confirmatory of malignant melanoma.

Lupus Erythematosus of Lid.—The patient shown in Figure 11 was a Negro woman, age 31. As shown in the photograph, there was partial involvement of the upper lid of the right eye and all of the lower lid. The latter involvement extended onto the adjacent skin; part of this lesion showed a scarred apigmented area with an erythematous, scaly border. The cilia were

Fig. 12.—Lupus erythematosus of the conjunctiva as described in the text.

absent, and the conjunctiva was not involved. The left eye was unaffected and there were no lesions of the disease elsewhere.

Lupus Erythematosus of Conjunctiva.—This is shown in Figure 12. The patient was a Negro woman, age 50. Lesions of lupus erythematosus were on the scalp, upper lip, and, as shown in the photograph, on the upper lid of the left eye, and below the lower lid, and involved all of the conjunctiva of the upper lid. The conjunctiva was raised, smooth, and bright red. At the ciliary margin there were crusted, raised, cornified lesions. The cilia were partly absent.

Delong and I [24] reported on lupus erythematosus of the eyelids, lid margins, and conjunctiva. The disease of the eyelids and adjacent skin has the clinical features that characterize chronic lupus erythematosus. The disease may be confined to one or both eyelids; it may be unilateral or bilateral, with, or without lesions elsewhere, notably the face and scalp. Affecting the eyelids the cilia are partially, or completely absent. The conjunctiva may, or may not be involved. Conjunctival involvement, without involvement of the lids or lid margins, is rare, in which event diagnosis would be difficult. Conjunctival involvement is presented as hyperemia, more likely, as extreme redness with velvet-like edema, or, as well defined, dry, white, atrophic areas, resembling the disease as it affects the buccal mucosa.

Lupus erythematosus of the lids and lid margins is not rare in the clinic at Wills Hospital. It is predominantly seen in the Negro.

Lymphoma.—Involvement of upper and lower lids of both eyes, of two years' duration, is shown in Figure 13.

The patient was a white man, age 65. The swelling of the lids was painless, not tender, noninflammatory, somewhat rounded, circumscribed, movable and rubbery to touch. There was a similar swelling on the hard palate, the surface of which was ulcerated. Both tonsils were enlarged and only the anterior cervical glands. A few small circumscribed nodules were present on the temple.

Fig. 13.—Lymphoma of the lids as discussed in the text.

Roentgenogram of the chest showed no abnormalities. The leukocyte and differential counts were within normal range.

Report on biopsy by Dr. Herman Beerman was probably lymphoma. The section showed dense infiltration with lymphocytes and some larger pale cells.

Melanosis of Conjunctiva.—There is a confusion of names in ophthalmologic literature in designation of such lesions. To wit, the nevus fusco-caeruleus ophthalmomaxillaris—the nevus of Ota. This is, essentially, according to Dorsey and Montgomery [25] a Mongolian spot located on the side of the face and involving the eye. Pariser and Beerman [26] likewise reported on the similarity of the nevus of Ota and persistent extrasacral Mongolian spot.

Reese [27-29] and also Greer [30,31] have notably studied melanosis of the conjunctiva. Their studies afford a comparison of such lesions with those of the skin.

Reese classifies melanosis of the conjunctiva as congenital and acquired. Of congenital nevi in children, 30% are nonpigmented; malignancy rarely ensues. Melanin may increase rapidly, especially at puberty, so that a previous inconspicuous lesion might appear. He advises removal of all nevi.

Acquired melanosis usually appears at middle age; it is termed by Reese as pre-

cancerous melanosis. It has a long period of many years in the precancerous stage, may remain stationary, or may progress to cancerous stage or may regress. It may be associated with lesions on the skin—notably, the face or eyelid—or it may be confined to the bulbar conjunctiva, or the palpebral conjunctiva with extension to the caruncle, the semilunar fold and lid margin. The lesion may be single or multiple of various size and configuration, dark brown, black or slate colored. The pigmentation has a fine granular appearance, and often shows spontaneous waxing and waning.

Histologically, it seems to begin in the basal layer of the epithelium and to show an intramucosal type of growth. He emphasizes exenteration in treatment of the malignant stage.

Of 134 cases of melanoma of the conjunctiva studied by Reese, 37 were precancerous melanosis, 64 were malignant melanosis, 28 were malignant arising from a nevus, and 5 were malignant; their origin from precancerous melanosis or from a nevus could not be determined.

The counterpart on the skin of what Reese calls precancerous melanosis has long been termed in dermatologic literature as melanotic freckle (Hutchinson), mélanose circonscrite précancéreuse (Dubreuilh). The aforegoing clinical features of the lesion on the conjunctiva, as described by Reese, are similar to its cutaneous counterpart as reported by Klauder and Beerman.[32] In the latter study its cancerous potentialities were not regarded as high as its occurrence on the conjunctiva as reported by Reese.

Greer classifies melanosis of the conjunctiva as benign, precancerous, and malignant. He regards conjunctival precancerous melanosis essentially as intraepithelial proliferation originating in the basal layer of the conjunctival epithelium. He regards it as analogous to Bowen's disease or to extramammary Paget's disease.

Greer's experience in study of melanosis of the conjunctiva differs from that of Reese. Of 153 pigmented lesions, 118 were nevi, 30 were malignant melanomas, and 5 were precancerous melanosis. Of the 30 malignant lesions, 3 showed histologic evidence of nevi origin, 9 showed such evidence of arising from precancerous melanosis, and 18 showed no histologic evidence suggesting their origin. Greer concludes that precancerous melanosis is a rare condition and does not always become malignant. Malignancy arising from a conjunctival nevus probably occurs more often than Reese has suggested.

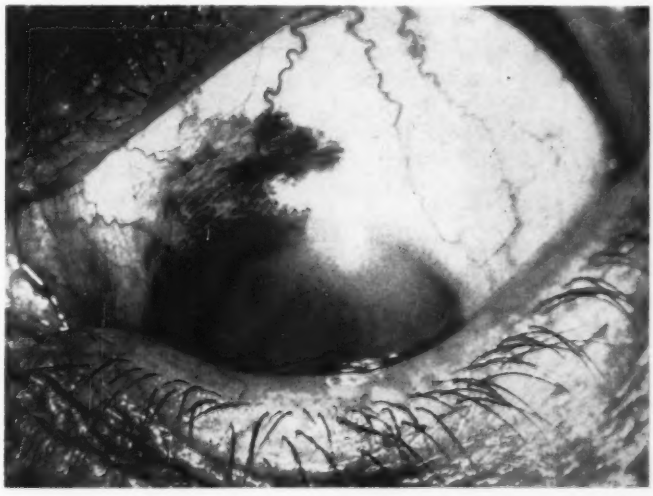

Fig. 14.—Benign melanoma of conjunctiva—precancerous melanosis, Reese (melanotic freckle, Hutchinson).

Fig. 15.—Malignant melanoma of conjunctiva, as discussed in text.

This experience of Greer is somewhat in conformity with the origin of malignant melanomas of the skin. The melanotic freckle of Hutchinson is a rare source of malignancy in comparison with its origin from the functional type of pigmented nevi.

Figure 14 shows a benign melanoma of the conjunctiva—precancerous melanosis, Reese (melanotic freckle, Hutchinson). The patient was a man age 56. He thought its duration was about ten years.

As shown in Figure 14, the lesion affected the bulbar conjunctiva at the nasal side of the upper limbus, extending upward. It was irregular in configuration and dark brown. The pigmentation was granular in appearance and was not uniform. Its upper part on the temporal side was much darker.

Figure 15 shows a malignant melanoma of the conjunctiva. The patient, a man age 49 came to the Ophthalmologic Clinic on account of hyperemia of the conjunctiva in March, 1953. He also presented a small pigmented spot on the palpebral conjunctiva on the nasal side of the limbus. He thought it was of a few weeks' duration.

He lapsed attendance until August, 1955. At that time he had a large mulberry-like growth extending from the inner canthus to the limbus. It was raised, localized, slightly movable, and brownish in color; its surface was vascularized.

The lesion was regarded as a malignant melanoma; the eye was enucleated. The

Fig. 16.—Unusual length of cilia—12 mm. as discussed in text.

pigmented lesion measured 22×22×25 mm., apparently arising from the conjunctiva. The sclera was not invaded, or were other regions of the eye. Histologic examination showed malignant melanoma.

Length of the Cilia.—Figure 16 shows a girl age 20 whose cilia measured 12 mm., longer than the average.

A casual search of text books and the literature failed to reveal information on the average length of the cilia. Piersol's "Human Anatomy" [33] only stated that the longest cilia (8 to 12 mm.) are in the center of the upper series. Danforth [34] writes: "It is claimed that they are slightly longer in women than in men." Texts on ophthalmology that were consulted discussed only developmental anomalies (as does LeDouble and Houssay) [35] and diseases of the cilia. Duke-Elder [36] writes: "Congenital hypertrichosis may manifest itself as an increase in the length of the number of the lashes. An increase in the size of the cilia (trichomegaly) is relatively common and may be a cosmetic asset, but as a rarity they may assume an enormous length (over 2 cm.; V. Michel, 1876; Gray, H., 1944, in a father and daughter). Hypertrichosis of the eyebrows and lashes may be familial (Marques, 1928)."

The aforementioned scanty information motivated measurement of the cilia of 250 persons including male and female in an age range from 4 months to 80. Measurement was made of the lashes of the upper lid and in the center where they are longest.

In children up to 10 years, including some infants, the length of the cilia varied

from 5 mm. to 11 mm. (a girl, age 4 years). The length in the teen age and the early twenties was fairly constant: 8, 9, and 10 mm. Only two persons in the 250 had a measurement of 12 mm. (2 girls age 19 and 20, respectively). In the late twenties and in the thirties the usual measurement was 6, 7, and 8 mm. About the age of 40, further decrease in the length was observed. In the forties and fifties, measurements of 5 and 6 mm. were common. Measurements of 3 and 4 mm. were common after the age of 60.

There were exceptions to the aforegoing statements. The margin of error in the length-age ratio was not statistically calculated. The smallest margin of error in this ratio was after the age of 60. The greatest margin of error was inherent in the 5 mm.-length category; here a greater variation of age was observed. Measurement of more than 6 mm. was not observed after the age of 50. There were three exceptions, a man age 79 and two women in their early fifties; one, whose cilia measured 8 mm., gave a history of familial tendency of long lashes. For only one other person in the study was there a similar history—a woman age 39, whose cilia measured 8 mm.

The possible difference in length of the cilia in the female compared with the male was not studied. The impression was gained that the cilia were longer in the female.

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Senile Purpura and Liver Disease

A Possible Relationship

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Bateman[1] (1836) is generally credited with the first description of senile purpura, which he had observed in elderly women. These lesions appeared

> . . . principally along the outside of the forearm in successive dark purple blotches, of an irregular form and various magnitude. Each of these continues for a week or ten days, when the extravasated blood is absorbed. A constant series of these ecchymoses had appeared in one case during ten years, and in others for a shorter period; but in all, the skin of the arm was left of a brown colour.

This brief but precise description was forgotten until near the turn of the century, when Unna,[9] as well as Pasini,[4] investigated the condition. The excellent article of Tattersall and Seville[8] gives evidence that senile purpura is constantly associated with an extreme degree of senile degeneration of exposed skin; the individual lesions are probably produced by minor external traumas acting on inadequately supported skin vessels. In the case report of Redslob and Bronner[6] the patient had classic senile purpura characterized by the usual location of the lesions, the atrophic state of the skin, the age and sex, and, finally, the recurrences. They emphasized ocular complications: recurrent retinal hemorrhages adjacent to the papilla and contemporary with the skin accidents.

Not all persons who have senile changes in the skin of the forearms suffer from senile purpura, and possibly other factors are responsible for the appearance of lesions in some persons and not others. It occurred to us that disturbed liver function might be one of the contributing factors. The intimate role of the liver in blood clotting has been appreciated for years. Thus, all evidence points to the liver as the chief source of fibrinogen and the sole

Bateman type of senile purpura.

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source of prothrombin. According to Lichtman, in frogs with extirpated liver the blood becomes incoagulable. The hemorrhagic tendency of patients with jaundice and the delayed coagulability of the blood in the cholemic state have long been recognized.

It has been shown that prothrombin concentration in the blood depends on the integrity of the liver and that liver damage is followed by corresponding impairment of prothrombin production.[7] In the classic study of Ratnoff and Patek [5] on the natural history of Laennec's cirrhosis, hemorrhagic phenomena were present in 99 of their 386 patients (excluding multiple symptoms this equals 25%). Seventy patients had epistaxes and thirty-three had purpura; of these, twelve patients had both symptoms. Other bleeding phenomena seen were bleeding gums in 17 persons, menorrhagia in 3, metrorrhagia in 3, and petechiae in 2. In addition to the hemorrhagic tendencies in cirrhosis, spider nevi, palmar erythema, and other signs of dilated vessels are usual.

Jones and Evans [2] point out that hemorrhagic manifestations during the course of hepatic disease are generally regarded as of serious prognostic import. Such instances are generally due to hypoprothrombinemia and indicate considerable parenchymal disturbance, as in acute hepatic necrosis and leptospirochaetosis. Jones and Evans add that purpuric hemorrhages may occur in comparatively mild hepatic diseases or in association with increased capillary fragility with or without thrombocytopenia. This form of purpura depends on tissue susceptibility and is not related to the intensity of the primary liver disturbance and, therefore, does not of itself indicate a grave prognosis.

In view of the above we sought out evidence of possible liver disease in a series of 20 patients with senile purpura (Table 1). Hematologic surveys were also made (Table 2).

These patients were not selected except that they were all derived from the Tulane Dermatology Outpatient Service at Charity

TABLE 1.—*Hematologic Tests* *

Case No.	PT	Hb.	RBC	PCV	ESR	WBC	MCV	MCH	MCHC	Th.	CT	CRT	BT	RL
1	11.8	13.5	4,400,000	42	38	8,500	—	—	—	934,000	47	—	3	0
2	12	—	—	—	—	—	—	—	—	—	60	—	5	++++
3	15	13.5	4,200,000	—	—	—	—	—	—	196,000	3.5	—	3	0
4	14.8	—	—	48	—	8,700	—	—	—	Adeq.	12	—	3	0
5	12.8	—	—	—	—	—	—	—	—	—	—	—	1	0
6	12.8	14.9	4,500,000	43	20	9,600	92	32	34	274,000	4	90	2	0
7	13.6	13.2	4,500,000	38	50	5,500	84	30	35	286,000	8.5	30	2.5	+
8	13.8	14.7	4,800,000	39	36	6,900	91	35	37	236,000	13	30	4	0
9	0	14	5,500,000	43	28	11,400	—	—	—	Adeq.	6	—	3.5	0
10	12.8	—	—	40	4	10,000	—	—	—	Adeq.	7.5	30	2	0
11	12.2	18	5,100,000	40	15	11,900	—	—	—	290,000	8	30	4.5	+
12	0	—	—	50	22	5,500	—	—	—	Adeq.	3	—	3	0
13	12.6	13	4,000,000	41	0	8,700	90	29	32.7	244,000	11.5	60	5.5	0
14	13.8	17.7	6,000,000	54	4	6,700	88	30	34	212,000	10.5	60	3.5	0
15	12.4	14.6	4,700,000	42	44	7,200	86	29	32	190,000	7.5	120	3	0
16	12.4	16.3	5,700,000	50	12	8,300	—	—	—	—	10	—	2.5	0
17	—	—	—	13	10	9,800	86	29	34	222,000	9.5	30	4	+
18	14	15.7	5,300,000	46	41	9,500	87	26	32	214,000	14	30	4	0
19	27	7.2	2,500,000	22	8	16,600	86	29	33	102,000	11	120	2	±
20	13.2	14.9	5,100,000	44	1	9,100	—	—	—	—	5	—	5	±

* PT indicates prothrombin time in seconds (normal 13.5-14.5); Hb., hemoglobin, gm/100 cc. (normal, females 14.0±2.0, males 16.0±2.0), RBC, red blood cells cu. mm. (normal, females 4.8±0.6, males 5.4±0.8); PCV, packed cell volume, % (normal, females 42.0%±5.0%, males 47.0%±7.0%); ESR, corrected erythrocyte sedimentation rate, mm., (normal, females 13, males 7); WBC, white blood cells/cu. mm. (normal 5,000-10,000), MCV, mean corpuscular volume, cu. μ (normal 87±5); MCH, mean corpuscular hemoglobin, $\mu\mu$g. (normal 29±2); MCHC, mean corpuscular hemoglobin concentration, % (normal 34±2); Th., thrombocytes/cu. mm. (normal 150,000); CT, clotting time, min. (Lee-White method) (normal 6-10); CRT, clot retraction time, min. (normal 60-90); BT, bleeding time, min. (normal 1-3, Duke Method); RL, Rumpel-Leede tourniquet test.

TABLE 2.—*Liver-Function Tests* *

Case No.	Age	Sex	Race	LP	SB	SBP	CF	TT	TP	A/G	AP
1	64	M	W	0	0.7	0	0	4.9	8.93	4.09:4.84	5
2	61	F	W	0	0.82	1.4	+	9.2	8.95	3.88:5.07	4.16
3	63	F	C	0	0.96	0	0	3.78	8.7	2.54:6.16	12.2
4	57	M	W	0	1.02	2	0	6.9	8.1		4.2
5	67	M	W	0	1.76	6.5	0	4.2	5.72	4.58:1.12	0
6	67	F	W	0	0.74	1.5	0	1.33	6.96	4.46:2.5	4
7	63	F	W	0	0.92	0	0	30	8.70	5.1:2.9	3.05
8	74	M	W	0	0.77	6.5	+	2.8	7.13	3.0:4.31	9.5
9	73	F	W	0	0.41	0	+	7.9	7.2	4.0:3.2	2.9
10	77	M	W	0	1.10	13.6	qns	1.39	6.24	3.36:2.88	5.25
11	65	M	W	3FB	0.74	9.3	—	1.8	7.48	4.08:3.40	3.3
12	54	M	W	4FB	1.2	6.5	+	2.9	6.51	2.92:3.59	4.1
13	75	M	W	0	1.5	22.5	0	5.4	6.35	3.69:2.66	4.2
14	78	F	W	0	0.5	20	0	0.3	7.5	4.1:3.4	6.5
15	66	M	W	0	0.6	3	0	1.7	6.3	4.05:2.25	4.5
16	53	M	C	2FB	0.5	4.5	0	—	7.76	3.82:3.94	3.2
17	65	F	W	4FB	0.9	22	+	4	6.79	3.58:3.21	2.85
18	68	F	W	0	.55	—	+	4.6	5.71	3.55:2.16	3.5
19	79	F	W	0	1.88	48.5	+	4	5.36	3.25:2.11	0
20	58	M	W	3FB	1.0	12.2	+	1.5	6.80	3.09:3.71	1.09

* LP indicates liver palpable, (FB, fingerbreadths); SB, serum bilirubin, mg/100 cc. (normal 0.2-1.4); SBP, sulfobromophthalein retention, % (normal not more than 5% dye retention in 45 min. specimen); CF, cephalin flocculation (normal no flocculation); TT, thymol turbidity, units (normal 0.5); TP, total protein, gm. % (normal 6-8.0); A/G, albumen-globulin partition (normal 1.5:1); AP, alkaline phosphatase, Bodansky units (normal 1.5-4.0).

Hospital. On physical examination, 5 of the 20 patients had clinically enlarged livers. Half of the entire group of patients had sulfobromophthalein (Bromsulphalein) retention in excess of normal limits. Eight of the twenty showed abnormal cephalin flocculation tests, and five of the twenty patients had excessive thymol turbidity values. The albumin-globulin ratio was reversed in seven instances; the same number of patients had alkaline phosphatase value above the normal range. From the standpoint of liver-function tests, only two patients (Cases 6 and 15) were entirely normal as measured by normal values for Charity Hospital. In general, the hematologic tests gave normal findings, but here also there were deviations out of the customary range. Thus, the Rumpel-Leede test was strikingly positive in one case and abnormal in three others. The clot-retraction time was prolonged in two instances; the prothrombin time, in one.

We were fortunate in being able to obtain needle biopsy specimens of the liver from 10 patients. Nine of these disclosed abnormal findings, and one was normal in every particular (Case 15). Changes included scars in the liver parenchyma, fatty changes in liver cells, regeneration and owl-eye nuclei, collections of lymphocytes about the portal triads, focal areas of necrosis, pigmentary derangements, and full-blown cirrhosis in one (Case 12).

Skin biopsy specimens were obtained from all patients from sites which were involved by the senile purpuric process. Our findings were consonant with those reported by previous investigators, namely, advanced senile degeneration of the skin.

Summary and Conclusions

The hypothesis is advanced that senile purpura may bespeak underlying liver disease, in addition to the cutaneous senile degeneration which has long been recognized. In order to study this point, liver brackets and hematologic surveys were obtained in 20 patients who displayed senile purpura. Of this number, needle biopsy of the liver was done in 10; abnormal findings were noted in 9 of these.

The conclusion is drawn that in this particular sample an unusual degree of liver disease was encountered. The suggestion is made that the finding of senile

purpura warrants an investigation of liver function.

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Colloid Degeneration of the Skin (Colloid Milium)

A Report of Seven Cases

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Colloid degeneration of the skin is an uncommon condition characterized clinically by the development of pseudovesicles in light-exposed areas. Approximately 84 cases have been reported since the entity was originally described, by Wagner, of Leipzig, in 1866.[1] We do not believe the condition is as rare as the sparsity of reports would indicate. We have collected seven cases over a six-year period and have seen several other unreported cases at meetings held in this area. Hand[2] saw three Australian soldiers with colloid degeneration within a year, and Gilbert and Cox[3] found this entity in eight soldiers over a three-month period. This unusually high incidence was undoubtedly due to the amount of sunlight exposure of the Australian troops in Northern Australia, New Guinea, and Borneo.

Colloid milium is found most often in fair-skinned persons who are chronically exposed to sunlight. The ratio of males to females is about 4:1. The pseudovesicles of colloid degeneration occur chiefly over the dorsum of the hands, the face, and the neck. Some lesions may regress if light

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Fig. 1 (Patient 1).—Colloid milium lesions over the dorsum of the left hand.

Fig. 2 (Patient 4).—Pseudovesicles over the dorsum of the left hand.

Fig. 3 (Patient 5).—Low-power photmicrograph, showing circumscribed colloid masses.

exposure is reduced, and in some instances they are severer during the summer than the winter.[3-6] The eruption is usually asymptomatic, but burning and itching may occur. Abnormal urinary porphyrins have been reported,[7] but this finding may be unrelated. The blood cholesterol has been elevated or high normal in several patients.[8]

The exact pathogenesis of this disease is not known, but sunlight plays an important role. Zoon et al.[9] showed that the colloid material is not a product of collagen or elastic tissue degeneration. They determined the amino acid content by filter paper chromatography and found it to be more closely related to serum proteins. This work has been confirmed by Becker and Wilson.[10]

Histologically, one finds circumscribed masses of hyaloid material in the papillary and subpapillary dermis. The colloid material stains eosinophilic on hematoxylin and eosin staining, but it is paler than the surrounding normal collagen. Fibroblasts are found in the homogeneous areas, and fissures occur secondary to fixation and dehydration. The epidermal changes consist of hyperkeratosis and atrophy. This condition may be differentiated histologically from amyloidosis only by special stains, e.g., Van Gieson's stain.[11-13]

No method of treatment has consistently given good results. Ascorbic acid (vitamin C) has been used by many investigators, but there are conflicting reports as to its value.[4,5,8,14,15] One must be careful when attributing improvement to ascorbic acid therapy because these lesions tend to improve at times without treatment. The best treatment seems to be avoidance of excess sunlight.

Comment

In general, this group of patients with colloid degeneration (Table) conforms with the over-all group found in the literature. The following differences may be noted:

1. All of our patients have been males.

2. The number of patients in this series suggests that colloid degeneration of the skin is not rare in this area. This high incidence in southeast Texas can be attributed to the long summers and large number of fair-skinned people in this area.

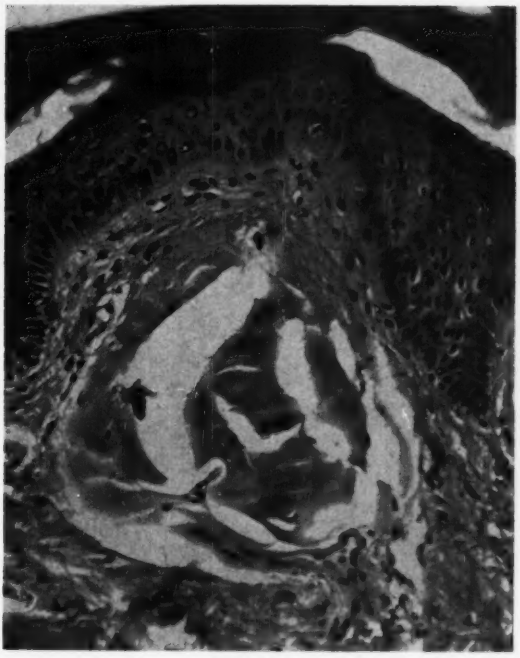

Fig. 4. — High-power view from Figure 3.

3. The dorsum of the hands was involved in every case. Whenever other areas were also involved, the hands were the worst areas.

4. If one hand was greatly different in severity, it was uniformly the left. Perhaps this can be explained by exposure to the sun on the left while driving an auto-

Data on Patients with Colloid Degeneration of the Skin

Patient No.	Age	Sex	Complexion	Occupation	Auto Driver	Location of Lesion	Duration of Lesion, Yr.	Urine Porphyrins	Blood Cholesterol, Mg. %	Other
1	33	M	Fair	Rancher	+++	Dorsum of hands; worse on left	15	None	389	Better in winter; purpura; burning on exposure
2	58	M	Fair	Truck driver	+++	Dorsum of hands	1	--	--	
3	53	M	Fair	Oil field worker	+	Dorsum of hands; worse on left	6	--	--	Purpura on hands
4	42	M	Very fair	Outdoor contractor	++	Dorsum of hands; worse on left	2	None	305	
5	47	M	Fair	Outdoor construction worker	+	Dorsum hands; helix bilat.; malar region	2	None	240	
6	53	M	Fair	Painting contractor	+	Dorsum of hands; worse on left; posterior neck	10	None	288	
7	37	M	Very fair	Sheet-metal worker	++	Dorsum of hands; worse on left; left malar region	12-15	None	168	Better in winter

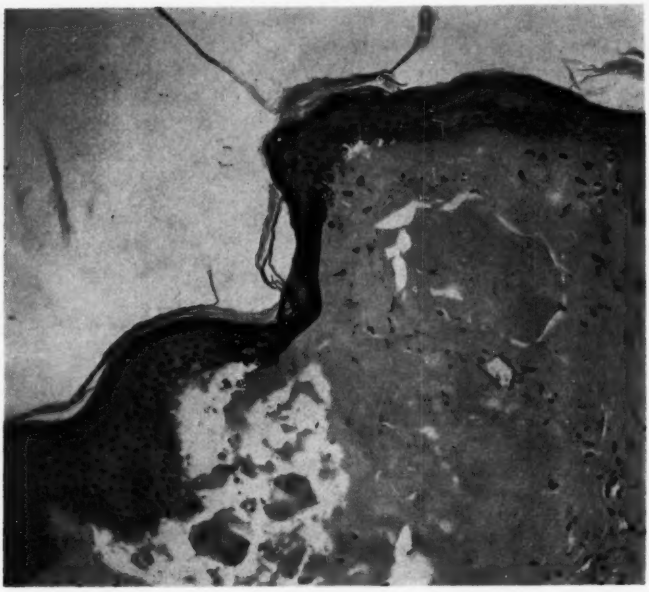

Fig. 5 (Patient 4).—Photomicrograph demonstrating extensive colloid degeneration.

mobile with the window down. One patient (No. 7) stated that he drove with the right hand and habitually gripped the window and roof of the car with his left hand. He also had involvement of the left zygomatic area during the summer months, and this might be explained on the same basis.

5. None of the patients improved while on ascorbic acid therapy. However, Patient 7 showed definite improvement six months after the ascorbic acid was discontinued. He was indoors during this entire period. It seems, then, that the patient's improvement can be attributed entirely to the avoidance of sunlight.

6. The cholesterol was elevated in four of the five patients tested. The significance of this finding remains to be determined. One must be cautious in drawing conclusions here, because of both the age group and the small total number of patients.

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Incidence of Dermatophytes in Cincinnati and Some Diagnostic Laboratory Procedures

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A short account of some figures will be given which we have accumulated during the nine years of the existence of the mycology laboratory in the General Hospital (Table 1). The overwhelming majority, or to be exact, more than 96% of all hair infections by Microsporum species are caused by Microsporum audouini. The absolute figures represent only a fraction of the total incidence, because even in the hospital certain cases, which are clinically typical and which show clear-cut fluorescence with Wood's light, are not cultured. This, indeed, refers in much higher percentage to cases seen in private practice, and one can only guess how many thousands of cases are seen by dermatologists, general practitioners, and pediatricians. One rather busy practitioner confesses to approximately 60 cases of M. audouini infection per year, and only the hope can be expressed that this is not the average number seen by the roughly 1,350 physicians in and around Cincinnati.

The diagnosis of M. audouini, as a rule, is not difficult. The hair fluoresces with typical greenish color. The direct examination of the hair generally reveals very numerous spores surrounding the hair shaft, and after four to six days one sees a transparent colony appear, which has feathery edges, which we always compare with spun glass, and after a few more days the back turns slightly salmon color. The mount reveals almost always an absence of macroconidia, and the microscopic diagnosis practically is made by exclusion, because one does not find diagnostic elements except for racquet hyphae and rare microconidia in thyrses.

In the hundreds of strains that we have examined, we had only two which had typical and numerous fuseaux of the Microsporum group, but no special clinical symptomatology was attached to the two specific strains, and we must just consider it a variation of nature. In the presence of numerous fuseaux, one may be hesitant to call the culture M. audouini, and then the rice medium is extremely helpful for making this decision. The rice medium actually is no rice medium but a few grains of rice, which are sterilized with a small amount of water in the autoclave; if one puts on the surface of this rice the culture in question, then one will get with M. audouini, only a brownish-rusty discoloration without development of any aerial mycelium, whereas, in the two other Microspora an abundant white to grayish mycelium develops.

As a rule Microsporum canis and Microsporum gypseum, in addition to other char-

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TABLE 1.—*Dermatophytes Isolated in Laboratory of Mycology, Cincinnati General Hospital, 1950-1958*

M. audouini	1,363
M. canis	43
M. gypseum	9
T. mentagrophytes	100
T. rubrum	154
T. tonsurans	48
T. violaceum	2
T. schoenleini	17
E. floccosum	11

acteristics, have pigment, but pigment can be exceptionally absent and the morphologic characteristics can be quite misleading, and one may feel very happy in the mycologic laboratory to be able to verify one's impression on rice medium.

M. canis, as you can clearly see, is no numerical problem in our community. The same can be said for M. gypseum, especially since the few cases which we have isolated in part came from pet monkeys and other pet animals. M. canis has become somewhat more frequent in the last few years, and in every single case we got the history of kittens in the family. As far as it was possible to examine these kittens they were uniformly infected and can be assumed to be the source of the human infection.

There is still considerable confusion, especially in the European literature, about the proper classification of the dermatophytes. We feel that Emmons [2] has done an enormous service to the teaching and understanding of the dermatophytes by his simple grouping of a handful of easily understandable names into three groups; these are Microsporum, Trichophyton, and Epidermophyton; this has eliminated the countless and confusing previously used names. The pendulum, however, swings occasionally in the opposite direction, and lately Paldrok has suggested to reduce all three species of dermatophytes to one genus "Microsporum," but we have never found anybody who would be persuaded to do so except for the author himself.[5]

There is little doubt that animals like kittens and puppies transmit the disease to man. From a hygienic point of view, there is very little excuse to keep pet animals, around children especially. If we look into the facts for spontaneous infection in wild animals as compared with domestic animals, one comes immediately to think of the unhappy role civilization has played toward natives. They paid a heavy price for their contact with so-called civilized man, in the form of alcohol, syphilis, and other derivatives of so-called civilization. It seems, from Table 2 and Table 3, that the animals

TABLE 2.—*Dermatophytes Isolated* *

Animals		
Wild	2,300	12=0.51%
Domestic	2,361	642=27.20%

* Adapted from Kaplan, W., et al.: *Ann. New York Acad. Sc.* 70:636-649, 1958.

could be equally resentful if they had the means of expression. In wild animals, of which approximately 2,300 have been examined by Georg and collaborators,[4] only a handful showed spontaneous infection with dermatophytes. In an equal number of domestic animals, however, 27% were shown to be infected. Without trying to break down the numbers in detail, it is very obvious that the major contaminating organism is M. canis, especially in dogs and cats. At this point we could mention that Trichophyton verrucosum, especially in New England, seems to be a considerable problem in the infection of farmers from cattle. It is universally true that infection of laboratory workers and other persons coming in contact with mice is considerable, since these animals, just like other laboratory animals, can be carriers of dermatophytes without exhibiting clinically recognizable features of disease.

One special feature, returning to the cultural characteristics, which permits differentiation in the Microsporum group, is the complete absence, for all practical purposes, of pleomorphism in M. audouini, whereas in M. canis and M. gypseum one gets grayish, fluffy overgrowth without spore formation, with regularity. This absence of pleomorphism in M. audouini can be of considerable help in questionable cases.

TABLE 3.—*Dermatophytes Isolated from Domestic Animals* *

M. audouini (dog)	1	T. mentagrophytes	52
M. canis	358	T. verrucosum	30
M. gypseum	84	T. rubrum (dog)	1

* Adapted from Kaplan, W., et al.: *Ann. New York Acad. Sc.* 70:636-649, 1958.

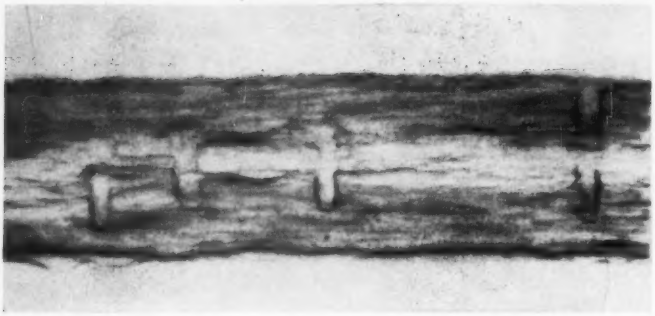

Fig. 1. — T. mentagrophytes perforates sterilized human hair in two to five days. T. rubrum does not attack the hair. Reduced 35% from mag. × 1,000.

Of Trichophyton, there are only two species which numerically play a role in our environment, and these are T. mentagrophytes and T. rubrum. While T. mentagrophytes occasionally is seen on the hair of the skull, this in T. rubrum is extraordinarily rare, but, if the skin is involved, then one frequently will be very eager to make the differential diagnosis and one may run into considerable difficulties.

Generally the pigment production of T. mentagrophytes is not very pronounced, most often it is a slight yellowish-tan color. The variants have an outspoken reddish pigment, and it can become extremely difficult to differentiate T. mentagrophytes from T. rubrum by simple morphological means.

Georg and Ajello[1] have developed a rather interesting method of differentiation which is somewhat laborious but which, if done properly, can be of some help. I refer to the method of exposing a sterilized hair to a liquid culture of T. mentagrophytes or T. rubrum. T. mentagrophytes regularly perforates the hair, whereas T. rubrum does not. The substrate in which this occurs is water enriched by a few drops of 10% yeast extract (Fig. 1).

Lately, griseofulvin has given us an excellent tool to differentiate the two fungi, because it is exquisitely fungistatic for T. rubrum, whereas T. mentagrophytes is much less sensitive to the antibiotic (Fig. 2).

One of the main features of the mycology laboratory is patience. If one can afford to wait a few days more with a culture, which seems to be a problem, the problem generally solves itself, either by development of specific structures, or by change of pigment and other similar features.

In the group of the faviform Trichophyton, it has been shown that especially T. verrucosum needs time for its develop-

Fig. 2.—T. rubrum is completely inhibited by 2.5μg. of griseofulvin per milliliter of media. T. mentagrophytes is much more resistant (inhibition with 20μg. per milliliter of media).

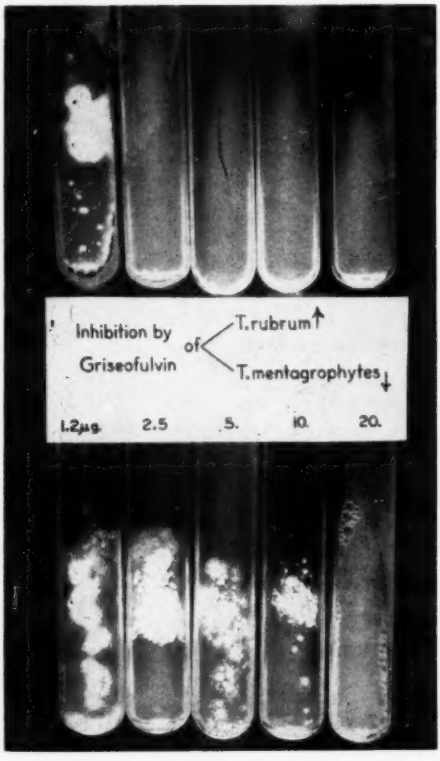

TABLE 4.—*Nutritional Patterns of Some Species of Trichophyton* *

	Thiamine	Casein	NH_4NO_3	NH_4NO_3 + Histidine
T. mentagrophytes	No stimulation	++++		
T. tonsurans	Stimulation	±		
T. schoenleini	No stimulation	++++		
T. violaceum	Stimulation	±		
T. verrucosum	Requirement	−		
T. megnini			−	++++
T. gallinae			++++	++++

* Adapted from Georg, L. K.[8]

ment, and, in general, one can state that it develops much better in the presence of the vitamin B complex than on ordinary media. Specific spores also may be formed in the presence of vitamin, whereas ordinarily the faviforms are characteristically devoid of fuseaux or microconidia. Table 4 shows rather strikingly what can be accomplished by the differentiation of organisms by nutritional tests. Some help in this respect can be also obtained in the differentiation of T. mentagrophytes and T. rubrum, and within the rosaceum group, which indeed is seldom seen in man and is mostly restricted to persons who have contact with chickens or turkeys. The differentiation of T. megnini from T. gallinae can be accomplished by the fact that T. megnini will grow only if in addition to NH_4NO_3 histidine is added to the medium, whereas T. gallinae does not require histidine.

Thiamine is of considerable help in differentiating several of the dermatophytes from each other. While T. verrucosum has complete requirements for thiamine alone, or in combination with inositol, T. schoenleini is rather independent of the presence of vitamins. A similar situation exists in differentiation of T. tonsurans from T. mentagrophytes and T. rubrum. The two latter ones are not stimulated by thiamine, whereas, T. tonsurans is readily recognized by its markedly better growth upon thiamine-containing media. Nicotinic acid can be extremely helpful in separating T. equinum from other dermatophytes.

Comment

The dermatophytes present a considerable problem from the social and economic point of view—much more so than from a medical one. Many of these infections have a spontaneous self-limited course, which especially in M. audouini, is said to be more than 30% of the cases. On the other hand, the patients ambulate from one physician to another with considerable excitement for the patient and the family, especially if contradictory advice is obtained or if school regulations forbid attendance, which frequently can become a considerable punishment for the parents. Up to now many dermatologists have epilated with regularity Microsporum audouini infections, if after a moderately short trial of conservative treatment no results were obtained. How soon, and how radically this will change with the introduction of griseofulvin is obviously too early to say.

In the Trichophyton group, the differentiation of T. rubrum, T. mentagrophytes, and T. tonsurans is also of considerable importance, because the easy response of T. mentagrophytes generally contrasts very much with the results obtained with T. rubrum and the frequently violent involvement seen in T. tonsurans.

I have humbly to acknowledge that without the dermatologists there would be no medical mycology, because they are the only group of specialists in medicine who can intelligently speak about fungi and attach meaning to most of them. However, if it seems mandatory for a physician to have

culture bottles in his office, it may be expressed that this should not give him the false illusion that he necessarily in every case can make the decision of the type of dermatophyte which is responsible for the disease which he observes in his patients. Everything has become more complicated with the deeper insight which we gain into cultural methods, nutritional requirements, pigment development of fungi, etc., and it is my feeling that sooner or later many of these cultures will have to be turned over to specialized laboratories if correct diagnosis should be the goal we are after.

The same may become true even if griseofulvin should be as good against some dermatophytes as it promises to be. If it should be shown that T. rubrum responds dramatically to griseofulvin, as we have any right to expect, and that T. mentagrophytes responds not so well, then it would be of considerable interest to establish the differential diagnosis with great certainty every time, since, undoubtedly, in addition to the already known toxic manifestations of the drug, others still will appear, and if one can assume that T. mentagrophytes could be cured by less hazardous measures, it certainly would be highly desirable to have this point completely clarified.

Summary

In summary, we can state that in our specific environment, the overwhelming majority of scalp infections is produced by Microsporum audouini; the absolute number of infections can be only guessed since only a small fraction is cultured in our laboratory; but, since we have between 150 and 200 positive cultures a year, the number in this general region must be simply enormous.

Microsporum canis has increased slightly and is almost always traceable to infected kittens. Trichophyton mentagrophytes and Trichophyton rubrum are almost equally numerous, which in other words means that T. rubrum has become more prominent than it used to be, and Trichophyton tonsurans has become a more or less steady inhabitant in this area, whereas, formerly it was considered a privilege of the South.

We can only hope that the introduction of griseofulvin will make the study of dermatophytes less and less necessary.

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Cholinesterases in Human Skin

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It is now generally accepted that many esterases exist in human skin. Lipases, α-esterases, "Tween" esterases, and specific and nonspecific cholinesterases have been described. Many of the methods are difficult to control and have given results of questionable chemical value. Many authors using these techniques have stated that the histochemical results were hard to evaluate. We wish to report our study of human skin with the Gomori technique, a simple method giving results that are histologically satisfying if not histochemically definite.

Method

The Gomori technique for cholinesterases was used.[1] Human skin was excised and placed immediately in cold 10% formalin in 0.9% saline. This was kept at 5 C for three to four hours. Simple formalin solution, acetate-formalin solution, and solutions of 15% sucrose and of 1% ammonium hydroxide in 10% formalin gave much poorer results. The stock solution was the same as that mentioned by Gomori.[1] This stock solution kept well at room temperature and butyrylthiocholine or acetylthiocholine was added before incubation. Twenty milligrams of substrate per 10 ml. of stock solution was employed. Incubation routinely was continued four hours at 37 C. At the first study of each new tissue, sections were removed at 2, 4, and 20 hours for judgment of the histologic results. Twenty hours adds intensity of reaction at the cost of specificity. The pH of the solution is about 6. If it is made more acid, the histologic results in general are poorer, although for intensely positive areas, such as end-organs and end-plates, the reaction is much sharper.

The inhibitors used were diisopropyl fluorophosphate (DFP) at $M/10^{-6}$ concentration, physostigmine (eserine) at $M/10^{-5}$ concentration, solution of dimethyl carbamate of (2-hydroxy-5-phenylbenzyl)-trimethylammonium bromide (NU-683) at $M/10^{-4}$ concentration,* and BW-284 at $M/10^{-4}$ concentration.† Pretreatment for half an hour with the inhibitor was employed originally, but later it was found possible to add the inhibitor directly to the substrate and obtain satisfactory results. With prolonged incubation, the inhibitors do not work so well. If the incubation can be kept short and still produce histologic results, these can be rendered meaningful by adding inhibitors to the substrate.

Findings

Excellent histologic results in biopsy specimens of human skin were achieved by this technique. The Table gives a comparison of results found in the nerve trunks, the end-organs (which include Meissner corpuscles, Vater-Pacini corpuscles, and neuromuscular end-plates), and the sebaceous glands of human skin.

In the nerve networks of the dermis, and in the sebaceous glands also, the reactivity of the acetylthiocholine substrate was

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Section of Dermatology (Dr. Winkelmann), Section of Pathologic Anatomy (Mr. Schmit), Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

* Dr. William A. Davis, Hoffmann-La Roche, Inc., Nutley 10, N. J., kindly supplied the NU-683.

† Edwin J. de Beer, Ph.D., Associate Research Director, Wellcome Research Laboratories, Tuckahoe 7, N. Y., kindly supplied the BW-284.

Degree of Cholinesterase Activity in Dermal Structures

Agents		Structures *		
Substrate	Inhibitor	Nerve Axons	End-Organs	Sebaceous Glands
Butyrylthiocholine	None	1	4	1
Acetylthiocholine	None	3	3	2
Acetylthiocholine	DFP	0	0	2
Acetylthiocholine	Eserine	1	1	1
Acetylthiocholine	BW-284	0	1	0
Acetylthiocholine	NU-683	0	0	0

* 0 indicates no reaction; 1, minimal definite reaction; 2, mild reaction; 3, moderate reaction; 4, strong reaction. Degrees 3 and 4 are histologically acceptable.

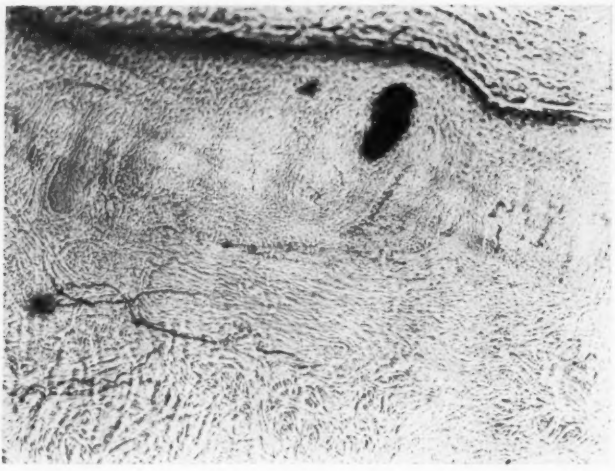

Fig. 1.—Nonspecific cholinesterase activity as demonstrated in the Meissner corpuscle in human hand; × 140.

stronger than that of the butyrylthiocholine; in the end-organ the reactivity of the butyrylthiocholine substrate was the stronger. It should be noted that DFP inhibited the reaction of the nerve in the dermis and of the end-organ but did not inhibit the reaction of the sebaceous glands. Eserine was only partially effective in all three sites. This could mean that the technique used—the addition of inhibitors to the substrate directly—was inappropriate. Yet the fact that DFP inhibited cholinesterase activity at known sites, such as the end-organs, indicates that it was acting in a typical manner. Its failure to inhibit the reactivity in the sebaceous gland region suggests that some form of nonspecific enzyme activity might be present which it could not affect.

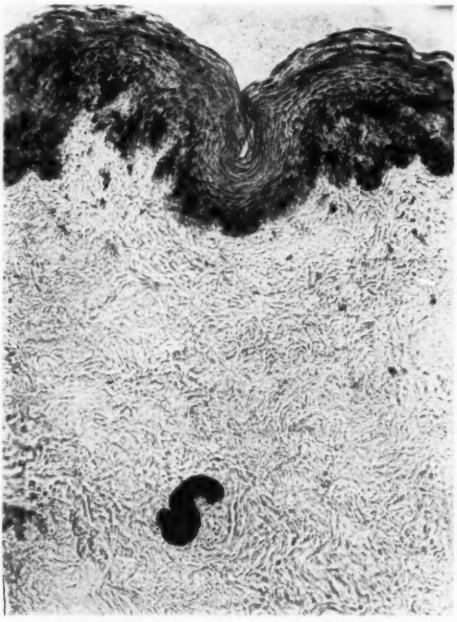

Fig. 2.—Nonspecific cholinesterase activity in the mammalian end-organ in the dog lip; × 130.

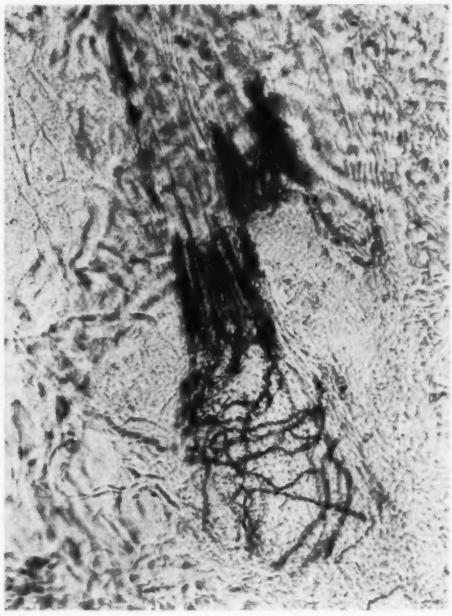

Fig. 3.—Nonspecific cholinesterase activity in the nerve network about the hair follicle, human skin; × 175.

It should be noted that NU-683 was effective in all sites of esterase activity. Similarly, it should be noted that BW-284, which supposedly is a specific acetylcholinesterase inhibitor, inhibited completely the reaction of nerves and of sebaceous glands, but only partially that of end-organs.

The data in the Table show that nonspecific cholinesterase is present in the end-organ of the skin and to some slight extent

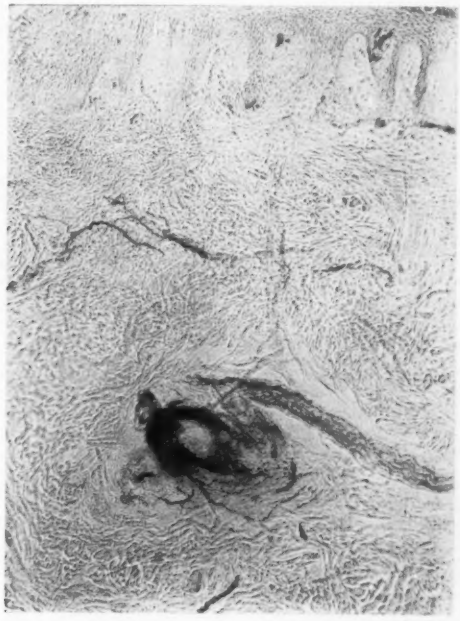

Fig. 4.—Cholinesterase activity in the glomus body of the human hand; × 90.

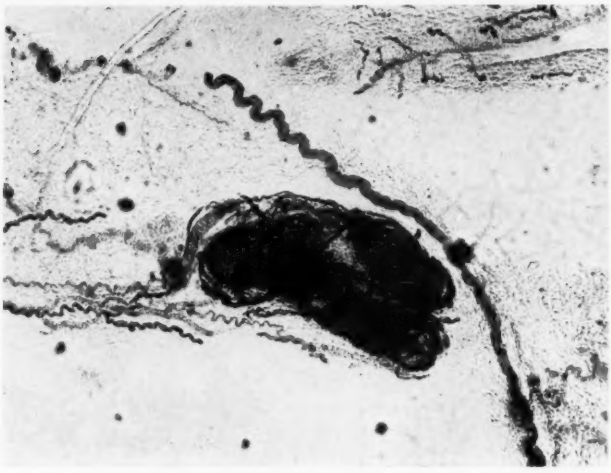

Fig. 5.—Acetylcholinesterase activity in the nerve network about the sweat glands. Note also the activity in the nerve trunk in the adjacent dermis; × 90.

in nerve networks. That the end-organ responds negatively with NU-683 and DFP and positively with BW-284 demonstrates that acetylcholinesterase is not present in it. The question of additional nonselective esterase activity is raised by the positive results in the sebaceous glands which were not inhibited by DFP. Partial inhibition of the sebaceous-gland reaction would be expected, as DFP has been shown to affect activity of nonspecific esterases such as α-esterase.

It was observed that some sites in which acetylcholinesterase could be expected afforded markedly positive reactions, and pseudocholinesterase reactivity was found in many other sites. The intense nonspecific cholinesterase activity of the Meissner corpuscle is evidenced in Figure 1. Some of the nerve network of the dermis is visible also. The inner bulb of the Vater-Pacini corpuscle is nonspecific cholinesterase positive. A similar intense reactivity is shown in the mammalian sensory end-organ in the dog lip (Fig. 2). The convoluted form of the end-organ, its position, and its intense nonspecific cholinesterase activity are all typical. An unexplained feature is the in-

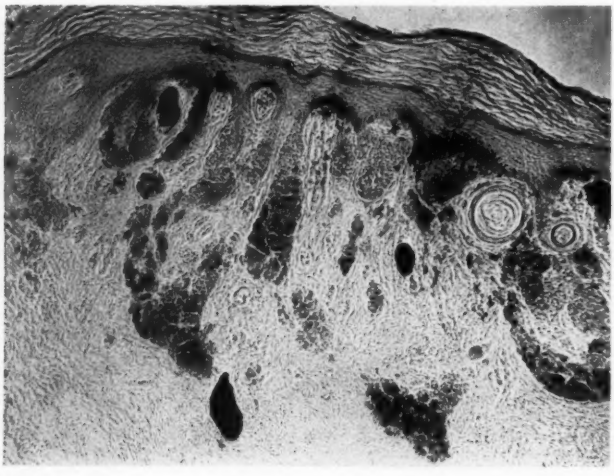

Fig. 6.—Cholinesterase activity in Meissner corpuscles in junction nevus of the human foot; × 85.

tense network about the hair follicles (Fig. 3). The acetylthiocholinesterase reaction outlines the double network of nerves which surrounds the follicles just below the point of insertion of the sebaceous glands. This intensity is present throughout the various stages of the hair cycle. Another similar network with marked specific cholinesterase activity is found around the glomus (Fig. 4). The positive reaction is in the nerve rete about the Sucquet-Hoyer canal. In comparison, the reactivity in the dermal network is mild. The density of the reaction around the glomus is similar to that shown by the sensory end-organs and by the neuromuscular end-plates.

Cholinesterase activity is diffusely present in the arrectores pilorum muscles of the dermis. No special localization to an organized nerve structure about the muscle was detected. A peripheral network of nerve tissue with positive acetylcholinesterase activity is found about the exocrine sweat glands (Fig. 5).

The Meissner corpuscle was studied as it occurred within junction nevi of the palms and soles. A representative area is shown in Figure 6. Note the intense nonspecific cholinesterase reaction of the end-organ as contrasted to the light-brown melanin pigmentation of the junctional nevus-cell masses. The nerve has not been disturbed structurally by nevus formation.

Comment

The simple Gomori technique has produced very satisfactory histologic results with the cholinesterase substrates. Due care should be given to the length of incubation and to the pH of incubation, with adaptation for the tissue and the species from which it has been taken. We agree with Coupland's [2,3] statements that pH 6 is best for the rabbit tissue. We do not agree that pH 5 is best for human tissue; the reaction at pH 6 is perfectly satisfactory histologically with the Gomori technique. A more acid pH suppresses staining of the dermal nerve networks in dog, cat, and rat, as well as in man.

The inhibitors may be added directly to the substrate, provided the time of incubation is not extended beyond reasonable limits and the optimal pH for the tissue is utilized. As shown by Koelle,[4] the inhibitor NU-683 proves eminently satisfactory for the inhibition of nonspecific esterase activity. DFP was useful but left something to be desired in its ability to suppress the reaction about sebaceous glands. Its toxicity and lability also were disadvantageous. BW-284 likewise was slightly unsatisfactory because, although it is expected to suppress only acetylcholinesterase activity, in use it inhibited positive results in the sebaceous gland. Eserine was unsatisfactory under the circumstances of our study. If two-hour incubation or pretreatment methods are used, the results with eserine are satisfactory.

These data confirm the findings of others that nonspecific cholinesterase is present in the sensory end-organs of the skin.[5,6] Since both specific and nonspecific esterases can act upon acetylthiocholine, one would expect the end-organs to be darker if specific acetylcholinesterase is present. They are darker when the butyrylthiocholine is used. Conversely, acetylthiocholine gives a stronger reaction in the dermal nerve network and about the exocrine glands [7] and is found about the hair follicles [8] and the glomus canal.[9] Probably one or more other enzymes are present in these dermal structures. Koelle has postulated the existence of a thioesterase.[10] It is entirely possible, in view of the mild sebaceous-gland esterase activity, that a nonspecific esterase might be present.

Summary

The Gomori cholinesterase technique has been modified to permit its routine histologic use. The use of inhibitors within the substrate itself simplifies the technical details. A successful inhibitor for nonspecific cholinesterase is the dimethyl carbamate of (2-hydroxy-5-phenylbenzyl)-trimethylammonium bromide (NU-683). Nonspecific cholinesterase is distributed in all

sensory end-organs found. Possibly, non-specific esterases capable of reacting with thiocholine esters are present also.

Mayo Clinic.

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Fox-Fordyce Disease

A Report, with Endocrinological and Histopathological Studies, of a Case Which Developed After Surgical Menopause

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Most reported cases of Fox-Fordyce disease have occurred in females. The series of Shelley and Levy [1] is the largest to date and includes 14 females but only one male. Winkelmann, Kierland, and Montgomery [2] found 23 cases in males reported in the literature and added an additional 1 of their own. They believed a reasonable female to male ratio to be 10:1. Shelley and Levy also pointed out that most of the women were in the 13- to 35-year-old age group. The disease has never been observed before puberty [3] and only rarely after the menopause.

The present report deals with a patient who developed the condition shortly after a surgical menopause.

Report of a Case

A 44-year-old white woman first noticed transient, mild axillary itching when she was in her late teens. At age 39, three months after hysterectomy and bilateral salpingo-oophorectomy performed because of the presence of cystic ovaries, she began to break out with a pruritic eruption of the axillae, areolae, and pubic area. She was first seen in the Dermatology Department of the University of Michigan Medical Center on Aug. 11, 1958.

The patient's cutaneous eruption was follicular and papular in type. Some excoriations due to scratching were present, and there was slight pigmentation. No lichenification was seen. Some, but not all, of the involved follicles showed loss of hair. The patient stated that the itching became worse under conditions which caused increased sweating. The rest of the skin appeared normal.

On Aug. 11, 1958, the urine was analyzed for hormone excretion with the following results being obtained for 24-hour samples: follicle-stimulating hormone, 48 mouse uterine units; estrogens, 3 rat units; luteinizing hormone not detected*; total 17-ketosteroids, 10 mg.; 17-hydroxysteroids, 9 mg. Vaginal smears (Papanicolaou technique) were compatible with an anovulatory cycle. The local injection of 0.15 cc. of epinephrine (1:1,000 solution) revealed apocrine anhydrosis in the axillary areas. Shortly after this test, the patient noticed a prickling sensation around the site of the injection.

Histopathology.—A biopsy† specimen 1.5×0.5 cm. was fixed in Zenker-formol for 6 hours, followed by immersion in Zenker base for 16 hours. It was treated with 3% $K_2Cr_2O_7$ for three days and double-embedded in celloidin and paraffin. Four hundred fifty serial sections, 4μ in thickness, were made. Several slides were stained with hematoxylin and eosin. Other sections were stained with the periodic acid-leukofuchsin (PAS) and counterstained with methylene blue in a citric acid phosphate buffer at pH 5.8 or with the Masson-trichrome and Harris' hematoxylin procedure.

There was marked keratotic plugging of the orifices of hair follicles (Fig. 2*A*). The apocrine sweat-retention vesicle described by Shelley and Levy was clearly visible (Figs. 2*A*, *B*, and *C*). In an apocrine gland at the level of transition of the secretory portion into the duct, a large periodic

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*FSH was assayed in immature white mice after recovery by the kaolin-adsorption method. Urinary estrogens were extracted with benzene after acid hydrolysis and assayed by the Allen-Doisy method. The hyperemia response of the immature rat ovary was used as a test for luteinizing hormone.[4]

†As Shelley[5] pointed out, vesicles are demonstrated better when the biopsy specimen is taken immediately following the epinephrine test. In this patient, however, the biopsy was performed on the side opposite to that of the test.

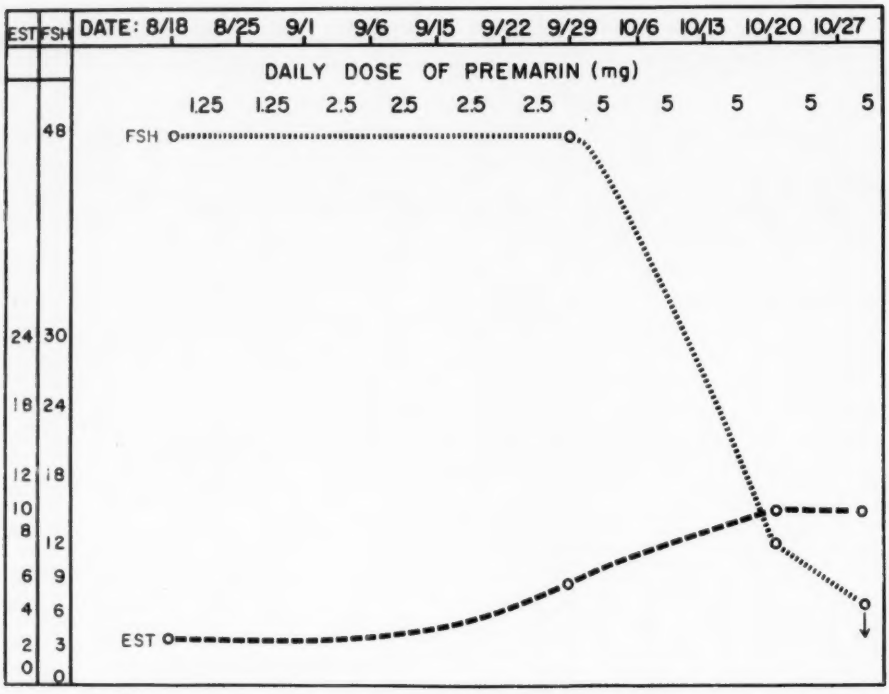

Fig. 1.—Changes in the 24-hour excretion values of urinary FSH and estrogens during 10 weeks of Premarin therapy. FSH is expressed in mouse uterine units and estrogens in rat units. The arrow indicates that FSH was not detectable at a level of 6 mouse uterine units or less.

acid Schiff (PAS)-positive, diastase-resistant cast was seen. In those sections stained with PAS-methylene blue, the secretory portion of the apocrine glands showed few PAS-positive cytoplasmic granules and little cytoplasmic basophilia (Fig. 2*D*). These conditions characterized only some tubules. Others were essentially normal, although most of them had a low epithelium. The basement membrane and myoepithelial cells appeared normal. There was a chronic inflammatory infiltrate around some tubules.

Treatment.—On Aug. 18, 1958, the patient was placed on oral, water-soluble conjugated equine estrogens‡ at a dose of 1.25 mg. daily. She experienced a decrease of pruritus during the first few days of treatment. Afterward, itching became worse again. For that reason, on Sept. 1, 1958, the dose was increased to 2.5 mg. daily. No substantial improvement was observed after four weeks on that dose. When the daily dose was raised to 5 mg. a marked decrease of pruritus occurred, although no striking change in the appearance of the lesions could be seen. The epinephrine test remained negative.

The hormonal changes which resulted from the estrogen therapy are illustrated in Figure 1 and summarized in the Table.

Vaginal smears taken on the same dates as those above indicated a progressive estrogenic action.

The 5 mg. daily dose was maintained until Dec. 15, 1958, and the pruritus remained under control during all of that period. On that date, it was reduced to 2.5 mg. daily and kept on the same level until Jan. 26, 1959, with only partial control of the pruritus. When the dose was reduced to 1.25 mg., a definite increase of the itching occurred.

Comment

Knowles[6] was one of the first to suggest an endocrine disturbance as being a possible factor in the etiology of Fox-Fordyce disease. Subsequently, Pick[7] reported two cases in one of which the oral administra-

‡ Premarin (Ayerst Laboratories, Inc., 22 E. 40th St., New York).

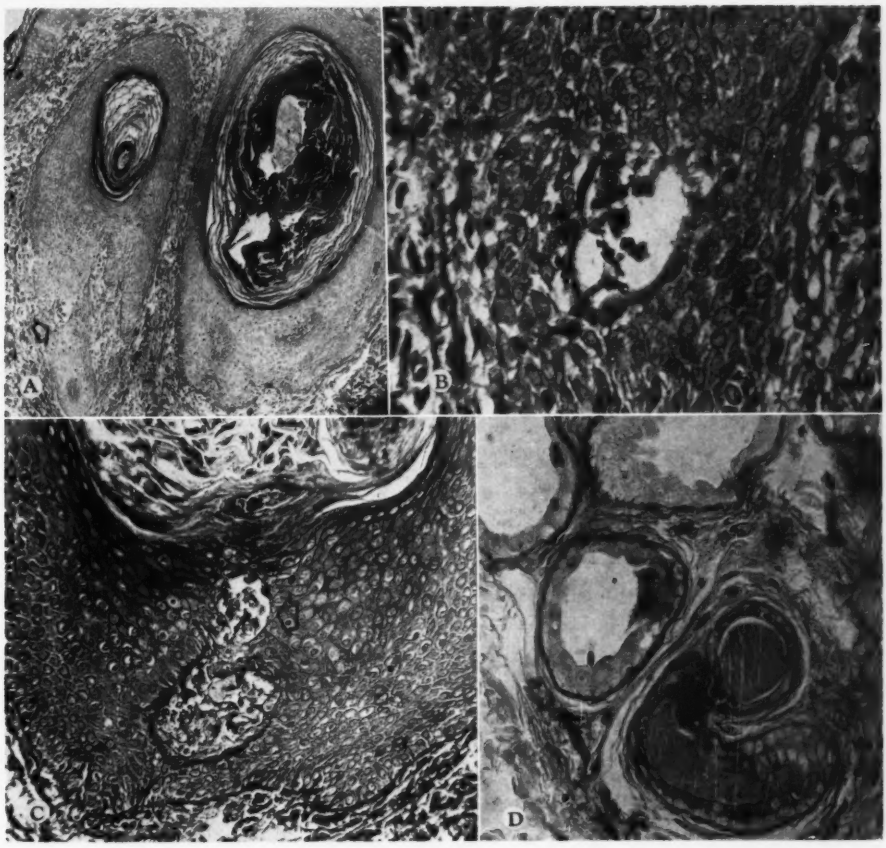

Fig. 2.—*A*, cross section of the epidermis, showing follicular plugging and the intraepidermal vesicle (arrow). Harris' hematoxylin and Masson; reduced about 45% from mag. × 105. *B*, the same vesicle as shown in *A*; reduced about 45% from mag. × 598. *C*, edge of a plugged hair follicle, showing another vesicle (arrow). Hematoxylin and eosin; reduced about 45% from mag. × 300. *D*, portion of an apocrine duct proximal to the secretory segment filled with PAS-positive material. The secretory coils have only a few PAS-stained granules. The cytoplasmic and nuclear basophilia is present in the secretory cells, although it is more visible in the cells of the duct. PAS and methylene blue; reduced about 45% from mag. × 570.

tion of 2 ovarian tablets t. i. d. resulted in a decrease of the itching without any striking change in the objective symptoms. In the other patient, both ovarian and corpus luteum tablets were used with a similar result being obtained. The continuous use of ovarian tablets was necessary for the control of pruritus, as there was recurrence of symptoms upon cessation of treatment. In both patients the itching was usually worse during menstruation. Pick concluded that Fox-Fordyce disease was a systemic condition which affected the apocrine glands, perhaps involving a dysfunction of them. The usefulness of estrogen therapy was mentioned by Chatellier [3] in the "Nouvelle

Variations in Urinary Estrogens and FSH During Premarin Therapy

Date	8/18	9/29	10/20	10/27
FSH *	48	48	12	<6
Estrogens †	3	6	10	10

* Mouse uterine units.
† Rat units.

Pratique Dermatologique." Furthermore, Roxburgh[8] used 1 mg. daily of diethylstilbestrol orally in a 25-year-old patient and obtained immediate improvement. When the dose was reduced to 1 mg. on alternate days, the improvement became slower. He also thought that the disease might be based on some endocrine disorder.

The condition seems to improve with pregnancy. One of Pautrier's pateints became completely free of the pruritus with disappearance of the skin lesions while she was pregnant, but there was exacerbation of symptoms following parturition.[9] Cornbleet[10] observed marked improvement in a patient after she became pregnant. Like Pautrier's case, the eruption reappeared after delivery.

Netherton[11] described the association of Fox-Fordyce disease with hyperthyroidism and the relief of symptoms following thyroidectomy. Fox-Fordyce disease has been observed also in association with Addison's disease.[12]

Part of the above data seems to indicate that in some patients the improvement has been related to an increase in blood estrogens levels. In the case under discussion, the clinical improvement observed upon estrogen administration could be correlated with an increase in urinary estrogens, a definite inhibition of pituitary FSH, and changes in the vaginal smears indicating a high estrogenic action. Why estrogens are effective in Fox-Fordyce disease is unknown. Since no changes were observed in the clinical appearance of the hyperkeratotic papules, and the epinephrine test remained negative, it is unlikely that the action was on the process of keratinization at the follicular opening. The direct influence of estrogens or other hormones on the secretory activity of apocrine sweat glands has not been demonstrated. However, Rothman[21] has suggested that increased levels of estrogens in the blood would depress proliferation of these glands.

It is possible that improvement of the condition under estrogen therapy is related to the induced low FSH level. In some patients, pruritus is intensified just before the onset of the flow, when FSH reaches one of its highest values during the menstrual cycle.[3,13] If the control of the pruritus is dependent upon the inhibition of the FSH, perhaps the use of higher doses of estrogens, or a more potent estrogen, such as diethylstilbestrol,[14] would give better results. Our finding of normal 17-ketosteroids excretion values is in agreement with Brunsting's studies.[15]

In recent years, two groups have sought to interpret the symptoms in terms of the pathology of the disease. Shelley and Levy,[1] on the one hand, emphasized the epidermal changes, demonstrating the rupture of the intraepidermal portion of the duct with the formation of an intraepidermal vesicle. They believed this to be the specific diagnostic sign and suggested that this vesicle, with the extravasated apocrine sweat, is the stimulus for pruritus.

Winkelmann and Montgomery,[12] on the other hand, found an abnormal amount of free mucin in the cutis and in the degenerated apocrine tubules. They considered the presence of mucin to be responsible for the pruritus. However, Baer and Witten[16] observed, "The presence of mucin in the tissues to account for the pruritus requires further explanation if one considers that neither lichen myxedematosus nor localized myxedema are pruritic, and both contain mucin." The apocrine sweat-retention vesicle was clearly seen in several different areas of the biopsy specimen studied. It also seemed to us that the presence of such a histological lesion is the more logical explanation for the pruritus.

The changes observed in some of the secretory cells (reduction of basophilia, scanty and small secretory granules, low epithelium) have already been seen in normal postmenopausal women by Ito and Iwashige,[17] Montagna,[18,19] and Montes and Baker.[20] It seems, then, that they would represent involutionary changes rather than being related to the Fox-Fordyce disease.

Summary

A case of Fox-Fordyce disease which developed after surgical menopause is reported. Histopathological study revealed the presence of the apocrine sweat-retention vesicle. The control of the pruritus upon estrogen administration could be correlated with an increase in urinary estrogens and a definite inhibition of the pituitary FSH.

Dr. Samuel J. Behrman and Dr. Gardner M. Riley directed the hormonal studies. The photomicrographs were taken by Mr. George Hess.

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Specific Systemic Antibiotic Therapy in Familial Benign Chronic Pemphigus

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Familial benign chronic pemphigus has long resisted the efforts of clinicians and pathologists alike to learn more regarding its precise nature. Unrecognized until a short time ago, it has attracted an uncommonly large number of names, as well as theories regarding its causation. The most recent students of this disease agree that it is an independent entity in which localized areas of epidermis show an inborn abnormal fragility.[1,2] Such epidermis is unable to withstand frictional forces as rubbing, scratching, adhesive tape, shoulder straps, or even tightly fitting collars.

It is our feeling that a second major pathogenic factor is bacterial infection. Although bacteria were demonstrated in some of the earliest cases of familial benign chronic pemphigus described twenty years ago,[3] it has never been the sense of the literature that familial benign chronic pemphigus is a pyoderma. In our opinion, however, a reexamination of the published cases lends much support to the view that familial benign chronic pemphigus is an unusual recurrent familial type of pyoderma. The following arguments may be marshalled in favor of the view that bacteria may initiate the lesions in familial benign chronic pemphigus.

1. The clinical appearance of familial benign chronic pemphigus is that of a pyoderma. The superficial serous crusting often seen in these patients mimics impetigo contagiosa, just as the bullae, flaccid or tense, suggest a diagnosis of bullous impetigo. Finally, in the axillae, vegetative malodorous plaques may arise which are clinical duplicates of pyoderma vegetans. It seems unquestionable that, prior to the papers of Hailey and Hailey, some of these patients were considered to have recurrent pyoderma. We should now add that the pyoderma is on a genetically altered soil.

2. Staphylococci may be cultured from early unruptured vesicles of familial benign chronic pemphigus.[3,4] This is compatible with the view that these bacteria are playing a primary rather than a secondary role.

3. Familial benign chronic pemphigus responds rapidly to antibiotic therapy. Although topical antibiosis only is recommended by Hailey himself,[5] a host of case reports attest to the efficacy of such systemic antibacterial agents as sulfathiazole, sulfapyridine, penicillin, and the tetracyclines.[4,6-12] With a daily maintenance dose of 200 mg. of aureomycin, Salsberg,[10] for instance, was able to keep his patient in complete remission for over a year.

The impetiginous appearance of familial benign chronic pemphigus, in conjunction with its reported response to antibacterial therapy, should lead to the conclusion that familial benign chronic pemphigus may be a special familial patterning of impetigo. What has prevented this view from holding the field? The major deterrent has been the inconsistent, erratic, unpredictable response of this disease to systemic antibiosis. Such an erratic response could be due either to the fact that bacteria play but an inconstant occasional role in the pathogenesis of familial benign chronic pemphigus or that drug-resistant organisms are actually the responsible agents. In this latter event, casual dispensing of antibiotics would in-

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variably lead to erratic unpredictable results. Our clinical studies with three cases led us to believe that bacteria do play a common, significant role in initiating attacks of familial benign chronic pemphigus. Furthermore, we have found that only by using the appropriate antibiotic (as determined by antibiotic-sensitivity typing of the bacteria present) can we expect regular success in treating the acute attacks of familial benign chronic pemphigus. The following case report is appended as an example of our experience. In this case only one of eight possible antibacterial agents could be expected to be effective on the basis of laboratory studies. It can be appreciated thus that random administration of antibiotics would seven times out of eight be futile. Thus, specificity of action is not only a desideratum, but a necessity.

Report of a Case

CASE 1.—This 43-year-old white man has been under our observation and care for the past two years. For nearly 25 years, he had had recurrent attacks of dermatitis of his neck, shoulders, and axillae. The initial lesion had been noted on the shoulder under a strap used in moving furniture. However, the axillary lesions have been the most persistent, rarely disappearing, whereas remissions of the neck and shoulder lesions did occur. At the time of his initial visit to our office, he denied any familial history of dermatitis. His general health was good except for a history of diabetes, discovered one year prior to seeing us. General physical examination was negative except for the presence of dermatitic plaques about the neck, shoulders, and in the axillae. Occasional bullae were to be seen on the neck, but the changes in the axillae were hypertrophic, fissured, and vegetative in nature. A clinical diagnosis of familial benign chronic pemphigus was made and two biopsy specimens (neck and axilla) proved confirmatory. Colonies of bacteria were seen in the outer stratum corneum. These were best visualized with the Giemsa stain, appearing as blue coccoid bodies. The Gram stain revealed them to be Gram-positive.

During the subsequent two years this patient was treated by us during five distinct attacks. Topical treatment of varied sort gave indifferent results, but we should like to summarize the significant findings with systemic medication.

Attack 1.—January, 1957: Sulfathiazole, 2 gm. daily, 2 weeks; no effect. Penicillin, 600,000 units, I. M., $\times 4$; rapid dramatic clearing.

Attack 2.—March, 1957: Penicillin, 600,000 units, I. M., $\times 1$; no effect. Prednisolone, 20 mg. per day; no effect. Novobiocin,* 1 gm. per day; dramatic rapid clearing.

Attack 3.—Occurred one week after novobiocin was discontinued. July, 1957: Novobiocin, 750 mg. per day; dramatic clearing, with reappearance when dosage dropped to 500 mg. per day. Able to discontinue in October, 1957.

Attack 4.—May, 1958: Novobiocin; no effect. Penicillin, 600,000 units, $\times 2$; clearing. N. B.: Pneumococcus isolated from vesicles which proved to be penicillin-sensitive on laboratory testing. Novobiocin sensitivity unfortunately not determined.

Attack 5.—Severe sudden appearance of bullae on neck and innumerable fissures of axillae. November, 1958: Chloromycetin, 1 gm. per day for 5 days; no help. Kynex, 0.5 gm. per day, 5 days; no help. Acthar gel., 40 units, $\times 1$; no help. Triamcinolone, 12 mg. per day, 2 days; no help. At end of week eruption rapidly progressing. Novobiocin, 1 gm. per day; sudden clearing. N. B.: Culture of bullae showed a coagulase-positive micrococcus resistant to aureomycin, chloramphenicol [Chloromycetin], erythromycin, penicillin, streptomycin, oxytetracycline [Terramycin], and sulfisoxazole [Gantrisin]. It proved to be sensitive only to novobiocin. Thus, the clinical and laboratory report shows close correlation.

With sustained dosage of novobiocin (500 mg. per day), the eruption has remained under essentially complete control for three months. Recent examination of the healed areas has revealed a positive Nikolsky sign. Uninvolved skin of the chest, arm, and elsewhere did not show the Nikolsky sign. There was no bulla formation in the healed skin on the injection of 0.1% trypsin,[18] indicating a normal response to proteases.

Analysis of this patient's clinical course and response to therapy indicated that bacterial infection may play a role in producing attacks. Thus, the bacterial factor can become fully as important as the trauma factor. Either or both may be operative in a given case. In any event, effective management calls for bacterial culture, antibiotic-sensitivity typing, and subsequent employment of the appropriate systemic antibacterial agent.

Conclusion

Evidence has been presented to indicate that bacteria, as well as friction, may play a significant role in precipitating an acute attack of familial benign chronic pemphigus.

The familial benign chronic pemphigus patient may be viewed as a person having

* Supplied (as Albamycin) by The Upjohn Company, Kalamazoo, Mich.

large areas of genetically-determined fragile epidermis. In the affected sites, for example, the axillae and neck, bacterial infections may repeatedly occur. Our experience would indicate that these are best treated by specific systemic antibiotics. This requires antibiotic-sensitivity testing of the particular strain of bacteria isolated. Thus, successive attacks or different patients may demand totally different antibiotics.

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Visual Disturbances with Antimalarial Drugs, with Particular Reference to Chloroquine Keratopathy

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The use of quinacrine (Atabrine) for chronic discoid lupus erythematosus was discovered by Page (1951).[1] It had been used independently for the same purpose in Russia and some Eastern European countries since the publication of Prokoptchouk's work (1940).[2] Atabrine was soon found to be the most effective known treatment for discoid lupus erythematosus and a great deal safer than gold therapy. However, it has certain disadvantages in addition to the yellow staining of the skin, and other antimalarial drugs were assayed for their activity in controlling this condition. Chloroquine was shown to be equally effective (Goldman et al., 1953)[3]—in some cases more so—and had the additional merits of not staining the skin and of showing less tendency to produce troublesome drug rashes, such as the lichenoid dermatosis from quinacrine (mepacrine). Both drugs will cause gastrointestinal upset and a number of other toxicity signs if the dosage is at all high. Chloroquine, however, produces two rather distinctive types of side-effect—visual disturbance and depigmentation of the hair (Howell, 1957)[4]—which are reversible and believed to be toxic reactions rather than a manifestation of allergy or idiosyncrasy. Neither is seen during quinacrine administration, but an appearance of blue haloes around lights which was noted by workers engaged in the manufacture of quinacrine (Atabrine) during the late war years attracted the attention of Ida Mann (1947),[5] who noted a granular deposit in the superficial layers of the cornea when examined with the slit-lamp microscope, an effect which she attributed to direct contamination with the quinacrine dust in the works atmosphere. Apart from this instance, visual disturbance resulted from the use of antimalarial drugs has been paid little attention, although "Clark's Applied Pharmacology"[6] refers to blurring of vision as a possible sequel of chloroquine administration, but Leeper and Allende (1956),[7] in their evaluation of antimalarials in the treatment of lupus erythematosus, make no reference to this. On the other hand, edema of the superficial corneal layers, detectable by slit-lamp microscopy and associated with transient, if serious, blurring of vision, was reported by Reese (1946)[8] and by Chamberlain and Boles (1946)[9] in small numbers of the airmen taking quinacrine as an antimalarial in suppressive doses, and in these cases the manifestation does seem to have been one of idiosyncrasy and was so regarded by them. A comparable experience with amodiaquin (Camoquin) has recently been reported by Bleil (1958)[10] as part of a severe toxic reaction to the drug.

It would appear, therefore, that two distinct types of corneal reaction to these drugs are seen: the one, as in Mann's cases, associated with a gradual deposit of opaque substance in the superficial layers of the cornea, and the second, a more acute, congestive reaction resulting in edema as seen by the American observers. It is to the first of these that our attention has been drawn in patients under treatment with chloroquine, and a preliminary description of it appeared last year.[11]

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The Royal Free Hospital.

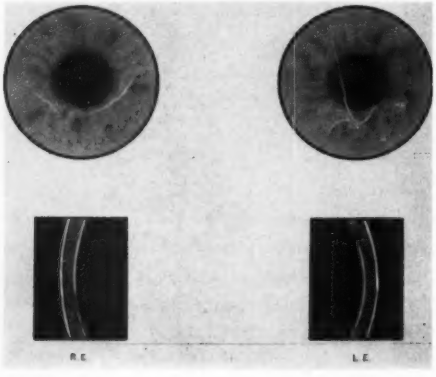
Chloroquine Keratopathy

The first case presented to one of us was in a woman doctor who had noticed a slight blurring of vision. The initial examination elicited excellent visual acuity and revealed no more than a small change in her refractive error with no sign of any pathological lesion. Both examiner and patient were content to accept the error as the explanation of her symptoms. A month or two later, however, when she complained that these had persisted and that she noticed as well that when driving at night car headlights appeared surrounded by colored haloes, the examiner was somewhat disturbed, since this is a prominent symptom of congestive glaucoma, and further examination was naturally directed to elicit signs of this condition, unlikely though it seemed in the circumstances. No such signs were forthcoming, but reexamination with the slit-lamp microscope revealed the presence in the corneal epithelium of a number of fine punctate deposits scattered generally over its central portion and aggregated below the pupillary area into a sinuous line (Fig. 1).

The presence of these changes offered an acceptable explanation of the blurred vision and haloes which she had noticed, since fine irregularities of the surface epithelium —most commonly edema in congestive glaucoma—are a familiar cause of such symptoms and with the slit-lamp microscope they may be seen with a fair degree of certainty. The origin of the disturbance in the chloroquine which she had been taking for her actinic dermatitis suggested itself from the similarity of the corneal appearance to that described by Ida Mann, in 1947. She showed clearly that there was an undoubted relationship between the corneal deposits and contact with quinacrine, although she believed that the condition arose from surface contamination of the cornea through exposure to powdered quinacrine in the atmosphere. The ophthalmological interest of our case lay chiefly in the demonstration that such an epithelial disturbance could be produced by drugs taken by mouth, since, although deposits in the corneal stroma which do not produce this visual disturbance have been seen from such metallic intoxications as mercury and silver; copper deposits are responsible for the appearance of the Kayser-Fleischer ring in hepatolenticular degeneration, and cystine crystals can be seen in Fanconi's syndrome, disturbance of the corneal epithelium from circulating drugs has not been described.

We were, therefore, interested to discover whether this first case was an isolated example, comparable with those described by the American workers and possibly following a period of corneal edema or if the condition arose frequently in association with chloroquine administration and was possibly the origin of the visual symptoms of which patients treated in this way sometimes complain.

Fig. 1.—Painting of slit-lamp microscope picture. Above: general appearance of corneal deposit. Below: optical section demonstrating situation of deposits in corneal epithelium.



Cases Examined

Some 30 patients to whom chloroquine was being administered in varying dosage and on different indications have now been examined: Twenty-three were suffering from lupus erythematosus; four were under treatment for actinic dermatitis, one, for largactil photosensitivity; one, for pityriasis lichenoides, and one, for clonorchis sinensis, and all but two of them have been seen during or immediately after a course of treatment lasting for several weeks to two years. To some degree these formed a selected group, for at the beginning

of the investigation at least they were those who complained of some visual disturbance.

The two exceptions were both cases admitted for observation at the beginning of treatment so early symptoms might be observed, especially if epithelial edema occurred as an early change. In both cases examination was negative, a finding which is perhaps of little value but which may be correlated with the absence in the history of the other cases of any symptoms suggestive of such a change in the early stages of treatment.

Symptomatology.—Of the remaining 28 cases, 19 complained of visual symptoms of one sort or another and 9 were asymptomatic. Examination of these cases, on one or several occasions, showed corneal changes in 22 and no or indefinite signs in 6. Of the 9 cases without symptoms, 5 showed definite signs of corneal deposits, and of the 22 with symptoms, no such changes were seen in 4. The complaint of some unspecified visual disturbance is evidently, therefore, not a reliable guide to the presence of the corneal changes which

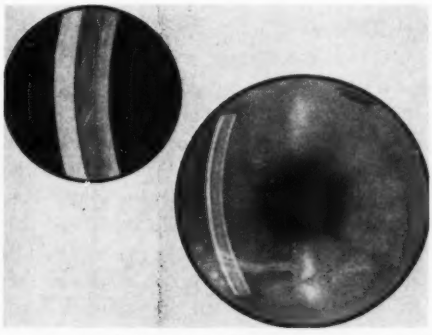


Fig. 2.—Painting of slit-lamp microscope picture. Late stage; general corneal appearance and (inset) optical section showing punctate and pigmented linear deposits in the epithelium.

are characteristic of treatment with these drugs. In the majority of cases visual symptoms of one sort or another arose either at the start of treatment or when the dosage was increased and appeared to result from intolerance of the drug. Such patients described "headaches and the vision closing in," "giddiness and the vision seems not to register," photophobia with nausea and a drugged feeling and momentary focusing difficulties in which (in one case) "the traffic dances for a few seconds." Focusing difficulties and transient blurring of vision are usually seen in hypermetropes in whom the general disturbance occasioned by incomplete tolerance of the drug leads to temporary impairment of the ciliary muscle, an effect which is familiar with other drugs and especially with the sulfonamides.

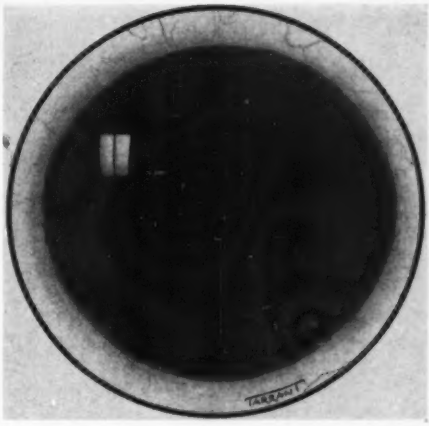
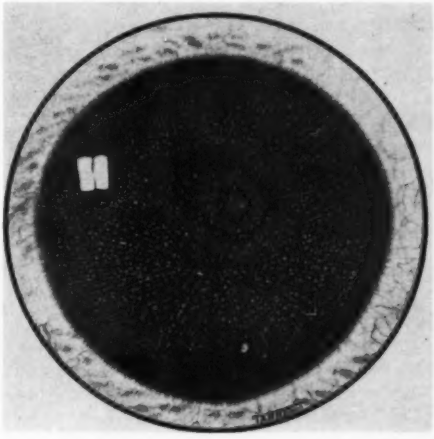


Fig. 3.—Painting of slit-lamp microscope picture. Intermediate stage; punctate and linear epithelial deposits.

On the other hand, spontaneous perception of colored haloes, the pathognomonic symptom of these corneal changes, has not appeared regularly in the history. It rose in the first case quoted but has been seen in only two others (both while driving at night), although a number of other patients have recognized as familiar the appearance of haloes when this diffraction phenomenon has been artificially produced by viewing a light source through a thin layer of lycopodium powder. This forms a most useful test. Patients with corneal changes commonly identify the appearance produced in this way with the blurred vision which they have experienced and when the light alone is presented to them see the somewhat fainter haloes which their own corneae produce.

Fig. 4.—Painting of slit-lamp microscope picture. Early stage; punctate epithelial deposits.



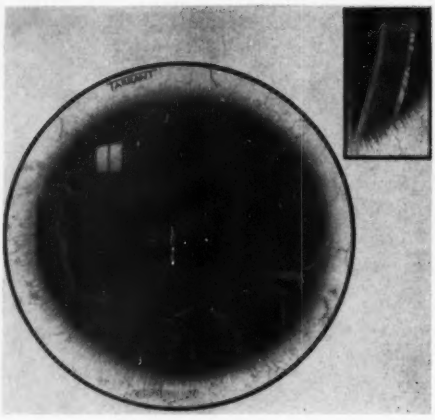


Fig. 5.—Painting of slit-lamp microscope picture. Punctate, linear, and limbal deposits.

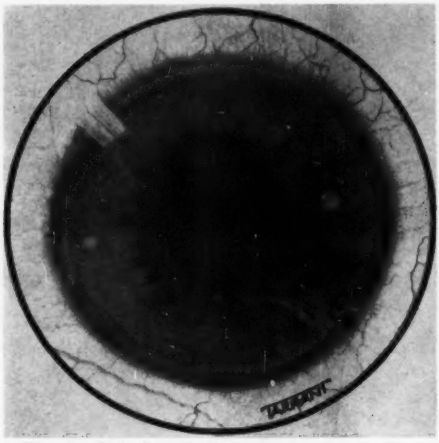


Fig. 6.—Painting of slit-lamp microscope picture. Epithelial macules and linear deposits.

Corneal Changes.—Examination of the corneae with the slit-lamp microscope showed several types of change in the 22 positive cases: In 9 of them the fine white dots and subpupillary line seen in the original case were apparent at the first examination (Fig. 2); 6 showed the widespread white dots in the epithelium with an appearance as though they were being aggregated into curved lines, concave upward and downward, their apices merging into denser lines just below the pupil, rather like lines of force in a magnetic field (Fig. 3), and in 7 there appeared simply a fine nummular stippling in the epithelium (Fig. 4). This last appears to be the early stage of the keratopathy, since it was seen to progress to the second type of change illustrated in two cases examined after a period of treatment and in a further case this change has been seen to progress to the complete picture with yellowish pigmentation of the subpupillary line. In addition, there have been three cases in which this fully developed change has been seen to regress after the cessation of treatment, in two of them with the complete absorption of deposits.

Apart from this picture, which we would regard as typical of the condition, there have been other associated epithelial changes in the form of irregular limbal deposits (two cases) (Fig. 5) and small maculae (five cases) (Fig. 6), sometimes in association with the typical picture and in other cases appearing separately but all of them in cases which at some time showed the typical change. In two cases pigmented maculae in the epithelium were seen in patients who had ceased treatment and in whom a linear deposit had originally been present.

Comment

The line which is seen in these cases is identical with an ophthalmological curiosity —the Hudson-Stähli line—which has for a generation been recognized to occur in association with long-standing ocular disease and in the normal eyes of old people but of which the precise significance is obscure. Its constant position below the pupil has attracted attention for a long time, and the suggestion was made by Koby, in 1930,[12] that this is determined by the action of the firm posterior margins of the eyelids, which come into apposition here when the lids close, thus tending in repeated blinking to push the loosely attached epithelium temporarily into folds in which particles aggregate. How far this explanation may be valid for the general instances of the condition is still in doubt, but among these cases it would seem to be an acceptable one.

The position of this line probably has some bearing on the appearance of haloes, since these will be most apparent where deposits are densest and the fact that the visual axis passes above this dense line may explain the unobtrusiveness of haloes in some patients when the condition is fully developed.

The gradual evolution of these changes during chloroquine treatment and the more

gradual recession seen in some cases when treatment has been discontinued make it fairly certain that they arise as the result of administration of the drug. The time needed for their evolution and the relationship to the dosage used are still uncertain, partly because regular observation of all of these patients, drawn from a wide area, has not been possible but also, we are sure, because individual reactions to the drug vary so widely. The earliest corneal changes which have been seen appeared only three weeks after treatment had been begun at a daily dosage of 300 mg., and these were more marked than in several other cases, treated for longer at higher dosage. In one case no corneal change was seen after two years' continuous treatment with a daily dosage of 600 mg.

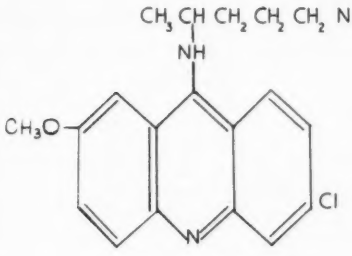


Fig. 7.—Quinacrine.

What the nature of the deposit is, what chemical radicle is responsible for its production, and how it reaches the corneal epithelium and remains largely confined to it are matters for further study. From the formulae shown (Figs. 7 and 8), it can be seen that the side-chains of quinacrine and chloroquine are identical but that the base of quinacrine is an acridine ring and that of chloroquine a quinoline ring. Although their therapeutic effects are so similar, there is no cross sensitization in hypersensitivity reactions to them. Recently another antimalarial, hydroxychloroquine (Plaquenil), has been introduced for the same dermatological indications, the formula of which is identical with that of chloroquine except for an hydroxyl group. The side-effects of

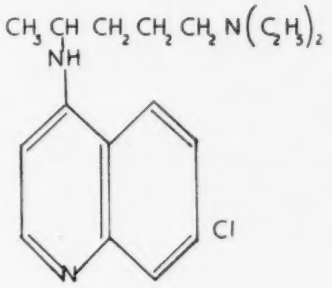


Fig. 8.—Chloroquine.

hydroxychloroquine are similar to those of chloroquine, but it is often better tolerated. On the other hand amodiaquine is a compound with a very different molecular structure (Fig. 9).

These antimalarial drugs are used in dermatology for lupus erythematosus and for actinic dermatoses. They have been and are still being used experimentally in a wide variety of other conditions but as yet without comparable results. Their beneficial effects have been thought to be attributable to a light-screening action, but while some experimental evidence exists to support this view, it is not proved. It is known that quinacrine has a strong affinity for the skin and its fluorescence enables it to be

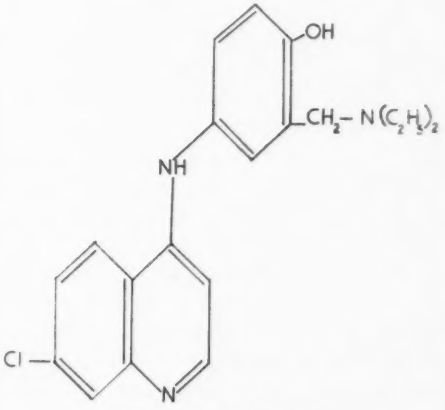


Fig. 9.—Amodiaquine.

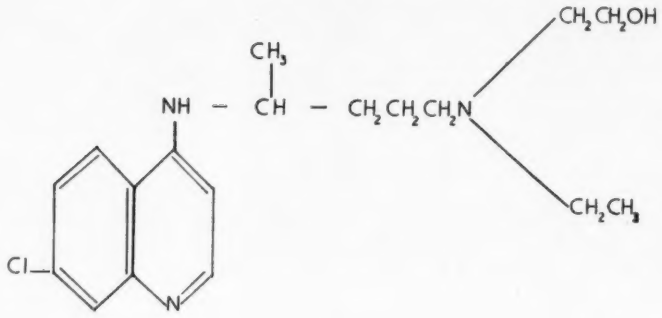


Fig. 10. — Hydroxychloroquine.

detected here, as well as in the hair and nails. The drug has been detected by this method in the sweat ducts of human skin six years after cessation of oral therapy (Hermann and Miller, 1952),[13] and it is known that there is a preponderance of the drug in the keratinous components and ether-soluble materials of the skin.

There has been no similar evidence that chloroquine has a particular affinity for skin or keratinous structures up till the present, apart from the clinical evidence of the hair-bleaching phenomenon (Marten, 1957).[14] It is only weakly fluorescent, and the methods for detecting it in tissues are not as sensitive as with quinacrine, but Brodie et al. (1947)[15] have described a fluorimetric method for estimating levels down to 10μg. per liter. Its fluorescence, however, seemed to provide a possible means of detecting it in the cornea, but examination of five cases with an ultraviolet-light source and quartz slit-lamp microscope failed to reveal any sign of fluorescence, this somewhat slender evidence suggesting that here, too, we are dealing not with the substance itself but with some organic product of it. If in man, as in rabbits, epithelial replacement takes place by a centripetal process from the limbus, the peripheral deposits seen in a few cases may well be evidence of the earliest stage in the evolution of the keratopathy, but these cases are few and the appearance of the majority does not support this view.

Very little is known of the metabolism of these antimalarial drugs in the body or of the biochemical basis for their therapeutic action. Silverman (1949)[16] has shown that quinacrine and chloroquine can inhibit the dephosphorylation of cocarboxylase by a phosphatase and the synthesis of thiamine in yeast cultures, while Kurnick and Radcliffe (1957)[17] found that quinacrine can form a complex with deoxyribosenucleic acid, which cannot be depolymerised by the enzyme deoxyribosenucleic acidase.

The dosage of chloroquine in dermatological conditions varies from 200 mg. to 1,200 mg. per day, although it is not usual to give more than 600 mg. per day. A loading dose of 600 mg. per day may be given for a week or more and gradually reduced to 200 mg. per day; alternatively the initial dosage of 200 mg. per day may be raised gradually until the dermatosis comes under control, and a maintenance dose of 200 mg. to 600 mg. per day may be continued for months or even years, although therapy may be intermittent according to the clinical indications. As a result, the dosage levels vary considerably in individual patients.

Duration of Corneal Changes.—The duration of the changes and their possible permanent effect upon vision is as yet uncertain: the earliest changes have been seen after a period of some three weeks' administration of chloroquine in a dosage of 300 mg. daily and the longest period for which corneal changes have been seen to persist

after the drug has been discontinued is, so far, two years. This last patient has been entirely unaware of any visual disturbance throughout the period of treatment, visual acuity is unimpaired, and the question of absorption of the deposits may be thought to be of purely academic interest, but in the example first quoted, that of the woman doctor, although objective signs in the cornea are now minimal two and one-half years after the cessation of treatment, she is still conscious of a faint blurring of naked lights. We should expect this disturbance to become less as time passes and the corneal deposits, which are now so faint, to disappear completely. They have not in her case at any time reduced vision as estimated by Snellen's test types and, indeed, such a reduction has been seen in only one of these cases, in which vision has fallen from 6/6 to 6/12. It is clearly not possible, however, to assume that permanent interference with vision does not occur, more especially if treatment must be prolonged.

Conclusion

Corneal changes appear in an unknown proportion of patients treated with antimalarial drugs in two forms: In one, where a transient edema produces acute, severe blurring of vision, they arise as a toxic reaction in susceptible patients; the second is characterized by insidious deposits of an opaque substance in the corneal epithelium which affect vision in a more durable fashion. In the case of chloroquine, at least, these appear usually to evolve in a regular fashion but may present as simple corneal nebulae.

We are indebted to Prof. A. W. Woodruff, of the Hospital for Tropical Diseases, and to dermatological colleagues at both the Royal Free and St. John's hospitals, whose cases have provided much of the material for this investigation. Sir Stewart Duke-Elder's interest has enabled some of the ocular observations to be verified at the Institute of Ophthalmology, and the excellent slit-lamp paintings which have made recording of the stages of the chloroquine keratopathy possible are the work of Mr. T. Tarrant, of the Department of Medical Illustration there.

Duhring Laboratories, University Hospital, Philadelphia 4 (Dr. Calnan).

Addendum

Since this account was prepared, subsequent observations have confirmed the presence of corneal changes in a large proportion of patients under treatment with these compounds in high dosage. They indicate, also, that additional retinal changes are to be expected, some of which produce more serious permanent visual results.

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Unusual Eruptions Associated with Internal Malignant Disease

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Many years ago, Erasmus Wilson asserted that "cutaneous medicine is a branch of general medicine of no insignificant importance," and there is no doubt that we dermatologists are pervaded by a particular pleasure when we recognize that the patient's cutaneous lesions are the signs pointing to some general disease, such as diabetes or tuberculosis. Especially we are alert to the possibility that an unexplained pruritus or that a trivial, nonirritating but persistent rash may be the first sign of malignancy, such as mycosis fungoides, Hodgkin's disease, or carcinoma of the colon. For example, in May of this year a man was referred to us with an extensive vesicobullous eruption, somewhat resembling Duhring's disease but mainly localized to the arm, thighs, and upper back, areas which had been in contact with sacks containing cement. Histological examination of an intact bulla showed that the rash was eczematous in character, and the eruption vanished completely three days after resection of a lung for a bronchogenic carcinoma.

It is not intended to discuss any of the well-known manifestations but to confine attention to cases which appeared to be distinctly unusual manifestations of malignant disease or their sequelae.

Report of Cases

CASE 1.—A married woman aged 32 was seen in the Deaconess Hospital on Feb. 6, 1957, at the request of the consultant physician, Dr. Batty. When admitted on Jan. 24, her skin had shown a dusky pigmentation with slight exfoliation, but by the time of examination she presented a pitiful picture (Fig. 1). There was general exfoliation, with many raw hemorrhagic areas, particularly around the natural orifices; the mucous membranes were completely denuded, and the skin of the hands and arms especially reminded one of a severe pellagra. She was obviously desperately ill and died six days later. Autopsy showed massive intra-abdominal lymphosarcomata, so entensive that the pathologists could not tell the exact site of the primary lesion. Her previous history was as follows.

She was originally admitted to the hospital in November, 1956, for investigation of edema involving the legs and the lower abdomen. Examination showed a tense distended abdomen with a palpable mass below the umbilicus; the spleen was palpable, and there were enlarged supraclavicular, axillary, and inguinal lymph nodes. X-ray revealed no abnormality in the thorax but demonstrated in the abdomen a tumor mass with ascites. A tentative diagnosis of retroperitoneal sarcoma was made, and

Fig. 1 (Case 1).—Widespread cutaneous lesions in woman with abdominal lymphosarcoma.

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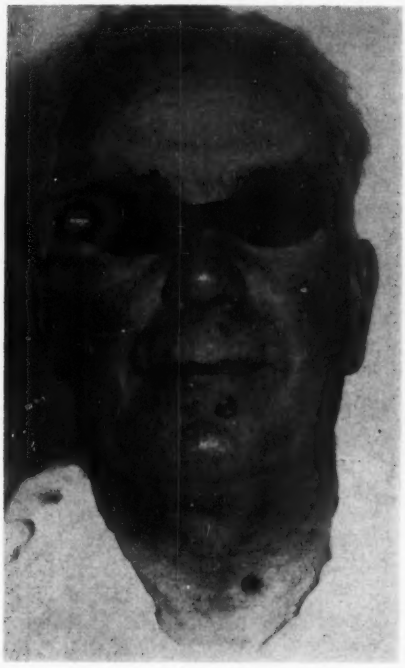

Fig. 2 (Case 2).—Cutaneous metastases on the brow, chin, and neck, secondary to dartomyosarcoma.

biopsy of an axillary lymph node revealed a lymphosarcoma. By the end of December, she had completed a palliative course of radiotherapy, and so on Jan. 4, 1957, she was practically symptom-free, though she had a large right-sided pleural effusion. She was readmitted three weeks later because of dyspnea and had not been aware of any cutaneous changes till then.

We do not know why this woman developed this severe fulminating exfoliative dermatitis. Was it due to toxic breakdown products resulting from the deep x-ray therapy, rather than to the lymphosarcoma itself, or was it a form of malabsorption syndrome, due to lack of pancreatic enzymes, folic acid deficiency, or disease of the bowel wall, caused by the tumors themselves?

Case 2.—On Jan. 22, 1958, a man aged 75 was referred for the diagnosis and treatment of three nodular growths. These had been present for six or seven weeks and were painless. Examination showed three tumors: one near the cleft of the chin, one on the neck to the left of the sternoclavicular notch, and a small one on the brow. These dusky-red nodules were sessile and of rubbery consistence and had a horny excrescence on the top (Fig. 2). His previous medical history indicated

Fig. 3 (Case 2).—Dartomyosarcoma after excision, showing nodular character of tumor.

that he had been a particularly healthy man but for 63 years had had a left inguinal hernia and 6 years previously noticed a hard lump in the scrotum perched on top of the hernia. In May, 1957, this lump suddenly ulcerated, and he was admitted to the hospital, where the tumor was excised (by Mr. Barron) (Fig. 3). Reference to his case notes of this period was made. The histological report (by Dr. N. McLean) on the neoplasm was as follows: "The dartos muscle of this scrotum is considerably hypertrophied and tumour can be seen infiltrating it—or possibly arising from it. The tumour varies considerably in its microscopic appearance. The two extremes are (i) areas composed of large spindle-shaped cells with hyperchromatic nuclei arranged in interlacing bundles and (ii) areas where pleomorphism is extreme and giant nuclei are frequent. No unequivocal myofibrils or transverse striations can be demonstrated by the use of a variety of stains, but the general nature of the cytoplasmic staining and the tendency to pleomorphism are more in favour of a myosarcoma than a fibrosarcoma. *Diagnosis*: Leiomyosarcoma of scrotum [Fig. 4]."

At this time the patient was otherwise apparently healthy, and no trace of metastases could be discovered in any organ, despite a thorough search. After his discharge from the hospital, he was seen once a month by the surgeon, but did not report after September, 1957, until referred to the skin department in January, 1958. The small growth on his forehead was excised for histological examination and as expected showed a highly active leiomyosarcoma (Fig. 5). The larger nodules were excised by the surgeon a few days later, and the pathologist, (Dr. Drennan), reported:

"The tumour as a whole is not circumscribed but infiltrates into its surroundings and the cells are more pleomorphic, the nuclei larger, with irregular chromatin patterns and occasional mitotic figures. The diagnosis is leiomyosarcoma. Comparison with the biopsy of the scrotal tumour shows that the latter is altogether more pleomorphic and malignant in appearance." All the sections were examined by four expert histopathologists, who agreed on the diagnosis.

At this time, x-ray of the chest showed doubtful signs of metastatic deposits at the base of the right upper lobe, but otherwise no clinical or radiological evidence of metastases could be detected. From then on, his condition deteriorated. On April 9, definite lung metastases were noted on x-ray; on May 16, he had a pathological fracture of the left humerus, and further tumors appeared on the skin of his scalp, back, and right arm. The liver was distended a hand's breadth below the costal margin and was hard and irregular. He died on June 8, with metastases in the brain, long bones, liver, and other organs.

Comment.—This case would seem to be an example of a dartomyosarcoma with metastases first appearing on the skin and later in other organs. We have not had the advantage of reading Hamilton Montgomery's [1]* recent paper on leiomyomata, and the most comprehensive article on this subject we know is that by Stout,[2] published in 1937. He states that the sources of plain muscle are: (1) the arrectors, (2) sweat gland muscles, (3) blood vessel muscles, (4) muscularis sexualis in the scrotum, penis, or labia majora, (5) muscularis mammillae et areolae, (6) similar musculature in the axillary and anal regions, (7) musculi cutis diagonales in the extremities, cheeks, scalp, and brow. He described 15 cases of leiomyoma and had found that 7 cases of malignant leimata had been found in the literature. Only two of these, however, had shown metastases as in the present case. One was a case of Devic and Gallavardin,[3] a patient who had had a subcutaneous nonadherent tumor of the buttock for some years, with sudden increase in size and eventual metastases in the kidneys, liver, lungs, pancreas, and thyroid. The other was a patient seen by Bartkowiak,[4] with a tumour of the skin of the foot which metastasized to the femoral lymph nodes, liver, and 11th dorsal vertebra.

Stout pointed out that a few cases have shown cellular variation and other microscopic changes, tending to suggest that they were malignant tumors, e.g., variation in relative size of cells with formation of bizarre and monster cell forms, swelling and vacuolation of muscle fibers, hemorrhages and necrosis, and increase in the frequency of mitoses, suggesting that these cases were *ipso facto* malignant. However, similar pictures are found in uterine leiomyomata, especially when degeneration is present, and do not necessarily denote malignancy. With no clinical signs of malignancy, they cannot be accepted as undoubtedly malignant tumors.

In this patient, the first sign of recurrence of the dartomyosarcoma was the appearance of nodular skin tumors of decidedly unusual aspect, and so the diagnosis depended on the histological examination of one of the tumors.

CASE 3.—A man, aged 51, was seen in June, 1951, for the diagnosis and treatment of an extensive dermatosis.

Apart from dysentery contracted in Iraq during 1919, he had been a particularly healthy man until 1947, when he was admitted to the hospital because of anemia (65% hemoglobin). There was no clinical abnormality, except that his liver margin was palpable. Sternal punctures showed an active somewhat macrocytic marrow but no suggestion of pernicious anemia. The stool benzidine was positive on several occasions; examination of the stools showed no pathogenic organisms and 20% total fat, of which 65% was split. Sigmoidoscopy was negative, as was a barium enema. Barium meal and swallow on two separate occasions revealed no

* This paper was written in the summer of 1958. Since then, Hamilton Montgomery and R. K. Winkelmann have published their article: Smooth-Muscle Tumors of the Skin, A. M. A. ARCH. DERMAT. 79:32-41, 1959. In their series of 33 cases, 1 example of leiomyosarcoma was observed, with no sign of recurrence 21 years after excision. They state, "Probably not more than a dozen cases of cutaneous leiomyosarcoma have been reported," and refer to the report by J. Levack and A. Dick (Cutaneous Leiomyosarcoma with Lymphatic Spread: A Report of 2 Cases, *Glasgow M. J.* 36:337-342, 1955). The second of these cases, they believe, would in the United States be interpreted as a rhabdomyosarcoma.

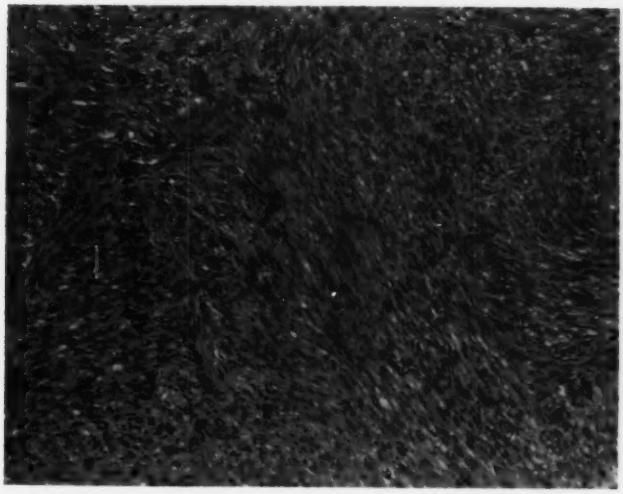

Fig. 4 (Case 2).—Dartomyosarcoma. Hemalum and eosin; × 75.

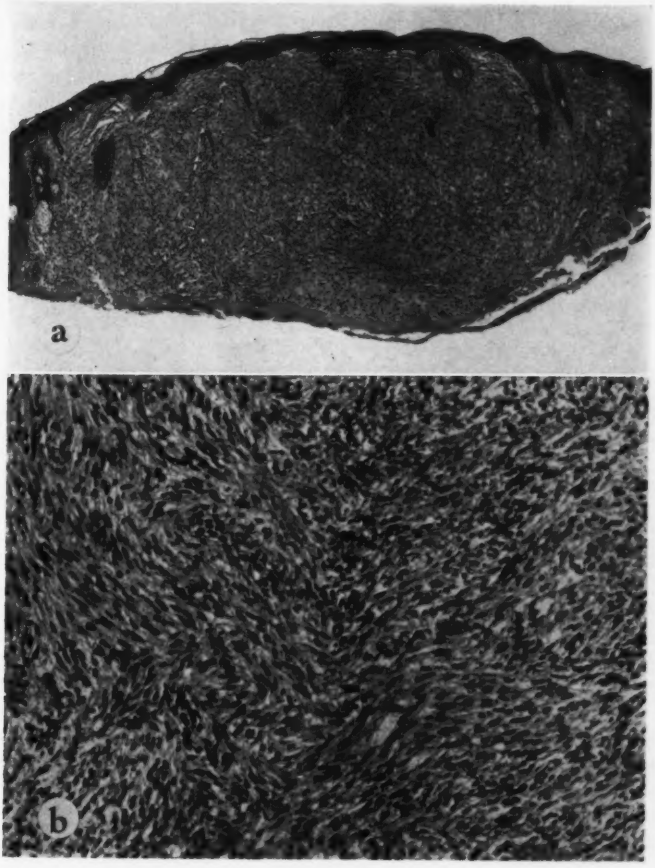

Fig. 5 (Case 2).—Dartomyosarcoma, secondary tumor of brow. Hemalum and eosin; (*a*) × 25. (*b*) × 130.

Fig. 6 (Case 3).—Patient with carcinoid and malabsorption syndrome. On the knuckles are lichenoid patches and on the wrists infective eczema.

abnormality. As no cause for his occult bleeding could be found, he was asked to report regularly as an outpatient, but he was next seen at the hospital as an acute abdominal emergency. A laparotomy revealed a carcinoid tumor of the jejunum, which had perforated, producing a peritonitis; he made an excellent recovery and was given a course of radiotherapy. At no time had he noted any flushing of the skin. For two years he kept well with no signs of recurrence, but in February, 1950, he developed a diaphragmatic pleurisy for which he received treatment with sulfonamides and penicillin. It was at this time that he first noticed a rash, in the form of red scaly patches on the shins. This persisted but could be controlled by simple local treatment.

After an attack of diarrhea, in early 1951, treated by succinylsulfathiazole (Sulfasuxidine), the dermatitis spread rapidly, and when seen in June, 1951, his appearance was strongly suggestive of a vitamin B deficiency. He had a raw red tongue, an angular stomatitis and cheilitis, and a dermatitis affecting flexures, such as the nasolabial folds, the ears, and the cleft of the chin. The scrotal skin was denuded, and there was a flexural eczema of the natal cleft and buttocks. From the mouth and flexures, Candida albicans was recovered. On the legs, arms, and hands there were low-grade septic infections with superficial ulceration and also pigmented areas and warty lesions, like verrucous lichen planus. The picture resembled a lichenoid drug eruption due to a heavy metal or quinacrine. No such drug was recovered from the urine, and his dermatosis was classified as a deficiency state (Fig. 6).

He was readmitted to the hospital in November, 1951, and suddenly developed on the posterior aspect of his left leg an eruption indistinguishable from linear lichen planus (Fig. 7). Many investigations were carried out, the most interesting finding being the appearance of the small intestine on radiographic examination. A skin biopsy specimen showed a picture compatible with lichen planus.

He received many forms of therapy. The most helpful local applications were chlorquinadol (Sterosan) ointment, iodochlorhydroxyquin (Vioform) cream, and methylrosaniline chloride (gentian violet). Intensive parenteral and oral vitamin therapy was administered but appeared to make little difference, and he was kept alive by repeated whole blood transfusions. He died in December, 1952, and autopsy showed no recurrence of the carcinoid, but in the small intestine there were extensive sections where the mucosa appeared to have atrophied and been desquamated. The cause of death was twofold: a bilateral bronchopneumonia and a thrombus of the abdominal aorta.

Comment.—This man had a nonfunctioning carcinoid tumor of the jejunum, and so the characteristic cutaneous flushing was conspicuous by its absence. Cardiac and pulmonary symptoms were not a feature of his case. Instead, he developed pellagra-like changes of the skin and buccal mucosa and also an eruption indistinguishable from lichen planus and somewhat resembling a drug eruption caused by a heavy metal or quinacrine. Such cutaneous changes

Fig. 7 (Case 3).—Linear lichen-planus-like rash.

were not referred to in the publications which have been consulted, e. g., the paper by Ritchie[5] has 232 references about carcinoid tumors, but no mention is made of any skin lesions except for the flushing and telangiectasis; Mattingly,[6] in reviewing the features of nonfunctioning and functioning carcinoid, reviewed 38 cases with no reference to this type of eruption. However, in the abstract of the discussion on the paper by Kierland, Sauer, and Dearing,[7] Dr. Shafer states: "It is of interest also that in some of the patients with this syndrome, particularly in those reported from Europe, pellagrinous changes have been observed. These are apparently secondary to the altered tryptophan metabolism and to a loss of nutrients from the gastro-intestinal tract." In this particular patient, with a nonfunctioning carcinoid, failure of absorption seems the likelier cause, and this seems to have been exacerbated by the administration of antibiotics and sulfonamides. Perhaps the radiotherapy given after the removal of the growth must bear some of the responsibility.

Summary

Three cases with unusual changes secondary to a primary malignant disease are described.

The first was a woman who had a widespread lymphosarcoma and who developed suddenly an exfoliative dermatitis.

The second patient had a dartomyosarcoma, which, six months after surgical removal, produced cutaneous metastases; the patient later died with disseminated secondary tumors.

The third, a case of nonfunctioning carcinoid of the jejunum, showed evidence of malabsorption, with an eruption like lichen planus and vitamin B complex deficiency.

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4 Walker St. (3).

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Postfuruncular Alopecia

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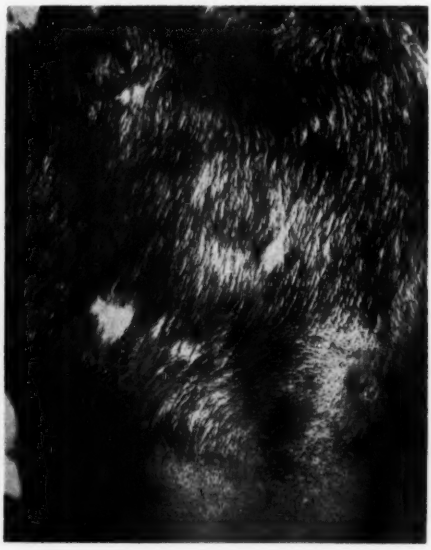
Pyogenic infections are somewhat commoner among the feebleminded than among normal people. The scalp is a frequent site of furunculosis. As a rule, the boil in this location runs its usual course in no way different from similar lesions on other parts of the body. Recently, among the 3,200 patients at the Pennhurst State School, Spring City, Pa., we have observed twenty-eight patients in whom furuncles on the scalp have been followed by temporary noncicatrizing alopecia about the site of the infection. Of these, we were able to follow 20 cases without interruption from the appearance of the boil to the complete regrowth of hair, and our observations on this group will be reported here. We have designated this temporary hair loss as postfuruncular alopecia.

The patients exhibiting this reaction to localized infection exhibited no physical or mental traits or laboratory findings that would distinguish them from their fellows. They were children and young adults varying in age from four to nineteen years. With one exception, all of the patients belonged to the white race. Three-quarters of them were males, suggesting the barber shop as a source of infection. In this regard, it is worthy to note that more than half of the girls involved wore their hair short. In 80% of the patients, the boil and subsequent hair loss occurred on the back of the head while, in the remainder, the lesions were divided equally between the temporal region and vertex.

At the beginning, there is nothing characteristic about the boil that will later lead to hair loss. A week or 10 days after the furuncle reaches its full development, sudden complete alopecia occurs about the inflammatory lesion (Figs. 1 and 2). The area of hair loss is round or oval in shape and varies from 2.0 to 5.0 cm. in size, with most of the patches measuring about 3.0 cm. in the longest diameter. At this stage of development, residual erythema may remain centrally at the site of the boil while about the periphery the normal pale color of the scalp is noted. During the time immediately before defluvium, the hair may be extracted manually with great ease and no discomfort.

Within a few days, all evidence of inflammation subsides, and there remains a bald area simulating alopecia areata. In two cases a small pitted scar was noted at the site of the crater of the boil, but in all others the smooth bald scalp exhibited no

Fig. 1.—Areas of alopecia at sites of previous furuncles. A new boil is present on the right side of the back of the head.



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From the Department of Dermatology, Pennhurst State School, Spring City, and the Department of Bacteriology, The Reading Hospital.

Fig. 2.—Same patient as in Figure 1 one week later. The boil has subsided and the nodule is surrounded by a zone of hair loss.

evidence of scarring. In the typical case, a uniform growth of fine hair is noted in about six weeks, and, two months later, the bald area is well-covered by approximately normal hair.

Variations from the above pattern occur. A patient may have several boils on the scalp without disturbance of hair growth, and then a furuncle appears which is followed by a surrounding zone of alopecia. In one case, there was definite thinning of the hair in the region about the boil, but complete loss did not occur (Fig. 3). Regrowth was uneventful. Later this patient had another localized infection of the scalp which followed the usual pattern of postfuruncular alopecia. In two of our children, regrowth of hair began about the periphery of the bald area and extended centrally, suggesting that perhaps the follicles about the edge of the lesion were damaged less than those situated closer to the boil.

Subsequent furuncles may be followed by the development of additional bald areas, so that a fairly large region of irregular baldness may result (Fig. 4). Sometimes a boil occurring just outside the denuded zone will delay regrowth of hair in the original patch and add another adjacent

Fig. 3.—Furunculosis of the back of the head. Only partial temporary hair loss followed these lesions but a later boil gave rise to typical postfuruncular alopecia.

Fig. 4.—Multiple boils have resulted in a large irregular area of temporary noncicatrizing alopecia.

area of alopecia. The time of regrowth of hair in both patches will then follow that expected for the second lesion. During these local changes the remainder of the scalp, skin, and nails are unchanged.

The causative organism in these patients was Staphylococcus pyogenes var. aureus, coagulase positive, phage type 80/81. The organisms recovered from these cases were often reported as sensitive to various antibiotics, but these agents were not employed systemically except in those with multiple concurrent boils. For most patients, the only therapy applied was an ammoniated mercury ointment.

An attempt was made to reproduce the alopecia in a lower animal. Staphylococci from two patients were grown in proteose peptone semi-solid agar, and after six days the cultures were centrifuged at 3,200 rpm for ten minutes. Supernatant fluid, 0.2 cc., from each specimen was injected subcutaneously into two places on the left side of a New Zealand white rabbit. At 48 hours two nodular plaques, each 1.0 cm. in diameter, had appeared at the sites of injection. Three days later there was slight hair loss over the anterior nodule, but a dry crust had developed over the other lesion. By the 10th day after injection of the toxin, there were more hair loss over the anterior nodule and a deep punched-out ulcer, 0.6 cm. in diameter, at the site of administration of the second bacterial product. During the next few days the ulcer became very dry while there was slight growth of the anterior nodule.

On the 15th day after injection there suddenly appeared a sharply outlined completely bald noninflamed area, 1.5 by 3.0 cm. in size, situated 4.0 cm. mesially and slightly cephalad to the anterior nodule. The smooth area of complete hair loss was located just to the left of the midline of the back (Fig. 5). At this time the site of the anterior injection had lost about 80% of its hair while only a small scaly area appeared at the point of administration of the second toxin. During the succeeding week, the anterior nodule became smaller and 90% hair loss appeared in an area, 1.5 cm. in diameter, about the lesion and extended backward in a partially bald band, 0.6 by 3.5 cm. in dimensions, to the site of the other injection.

On the 30th day after the administration of the toxins, regrowth of hair began in all the areas that had suffered alopecia. At first the hair was thin and silky, but by the end of 10 days the bald and the partially bald areas were completely covered with a good regrowth of hair.

An attempt was made to reproduce these observations in a black rabbit, using a

Fig. 5.—Partial hair loss over anterior (*A*) and posterior (*P*) sites of injection of staphylococcus toxin. Area of complete hair loss appeared just to the left of the midline of the back 15 days after injection of the cultures.

staphylococcus recovered from a third patient. We were unable to produce hair loss either at the site of injection or nearby. Since the epidemic of boils at the school has been brought under control, suitable cultures for further studies have not been available.

Next we tried to utilize the epilating effect of the toxins from the staphylococci in the treatment of tinea capitis due to Microsporum audouini. The phage type 80/81 Staphylococcus pyogenes var. aureus recovered from a furuncle of the head was cultured in fluid thioglycollate for forty-eight hours. The resultant growth was heat killed at 56 C for one hour and m-cresol, 1:1,000, added as a preservative. The killed suspension was then centrifuged at 2,200 rpm for fifteen minutes and the supernatant fluid discarded. The residue was mixed with hydrous wool fat and petrolatum in various concentrations and rubbed twice a day into the scalp of patients with tinea capitis. After six weeks of such therapy, the ringworm of the scalp in each case appeared to have spread when examined under the Wood's light.

Summary

A series of 20 patients is reported in which temporary hair loss resembling alopecia areata was noted after furuncles of the scalp. Alopecia occurs seven to ten days after the boil. Regrowth of hair begins in about six weeks and is complete in two months. Variations from this pattern are reported.

Toxins from one culture obtained from a furuncle when injected subcutaneously into a white rabbit caused partial hair loss about the site of injection and an area of complete hair loss, 1.5 by 3.0 cm. in size, situated 4.0 cm. mesial and cephalad to the site of administration. Regrowth of hair in all areas was complete. We were unable to reproduce these observations by injecting a culture from another patient into a black rabbit.

Bacterial toxins applied in ointment form to the scalps of children with tinea capitis were without favorable influence on the disease.

411 Walnut St.

Reports

Progress in the Biological Sciences in Relation to Dermatology

THOMAS B. FITZPATRICK, M.D., Oxford, England

During the next twenty years we may witness a veritable revolution in dermatologic treatment based on precise knowledge of the pathogenesis of various skin diseases. To bring about this change we need the assistance and guidance of basic scientists, and, indeed, in recent years, many scientists have discovered that the skin is a readily available organ in which basic processes can be directly observed.

This increasing interest in the skin as a fruitful organ for study was reflected in the "all-star" faculty that conducted a most successful course on "Progress in the Biological Sciences in Relation to Dermatology." The course, sponsored by the Postgraduate Medical School of Cambridge University, was held in Cambridge, England, from the 22d to the 27th of September, 1958. This was the first of a new series of courses presenting advances in the biological sciences to practitioners. There was a large attendance of 80 practicing dermatologists from all parts of the United Kingdom and from Holland and Denmark. The faculty and "students" lived together in Downing College during the entire course, where there was ample opportunity for exchange of information between dermatologists and the various basic scientists.

The success of any educational exercise depends on the quality of the faculty. The speakers consisted largely of well-known British scientists. Each presented a crisp, authoritative half-hour *précis* of their subject. Dr. Arthur Rook of Cambridge and the Regius Professor of Physic, Dr. J. S. Mitchell, who organized the course, wisely selected a dermatologist as chairman of each of the eleven half-day sessions. The chairman introduced the subject and led the discussion. A summary of the course prepared by Dr. Rook, which will shortly appear in the St. John's Hospital Transactions, is also presented below.

Submitted for publication April 27, 1959.

Session 1: The Melanocyte and Melanogenesis

Chairman, Dr. T. B. Fitzpatrick (Harvard)

Prof. J. D. Boyd (Cambridge) spoke on "The Embryology and Comparative Anatomy of the Melanocyte." He gave an account of the neural crest and of some of its derivatives; dorsal root ganglia, some cranial ganglia, the Schwann sheath cells of the peripheral nerves, some mesenchymal structures, including the leptomeninges, much branchial cartilage, odontoblasts and all pigment cells with the possible exception of those in the eye. He believed the evidence for the neural crest origin of the melanocyte to be conclusive. Von Recklinghausen's disease primarily involves neural crest derivatives other than melanocytes but abnormal pigmentation also occurs. Consideration of the distribution of melanocytes in various vertebrates suggested that those occurring in deeper situations cannot be concerned with reactions to radiant energy in or near the wave length of light; they may, therefore, have some additional function. The mechanisms controlling the migration of melanocytoblasts are largely unknown. Mature melanocytes have lost the power to move. Pigmentation possibly results from a qualitative change in melanocytes rather than from an increase in number.

Dr. P. C. J. Brunet (Oxford) spoke on Melanogenesis and gave a detailed but lucid account of the biochemistry of melanin formation and its control which provided much food for thought

on the possible mechanisms underlying familiar but obscure clinical phenomena.

Dr. J. I. Harris (Cambridge) speaking on "The Chemistry of the Melanocyte-Stimulating Hormones" gave a fascinating account of the steps in the elucidation of the chemical structure of corticotropin and MSH from pig pituitary glands. ACTH and MSH show striking clinical similarities and are perhaps produced by a common biosynthetic method. Dr. Harris wondered whether MSH has any physiological function in the mammal.

Dr. H. B. F. Dixon (Cambridge) completed the session with a lecture, "MSH and Corticotropin."

Session 2: Cutaneous Innervation

Chairman, Dr. J. T. Ingram

Dr. Graham Weddell (Oxford) speaking on "The Structure of the Skin" described studies on the cornea in man and laboratory animals, on the rabbit's ear, and on leprous skin which emphasize the lability in structure and function of sensory nerves. The information signalled to the central nervous system is relatively unaffected by cyclical changes which both tissues undergo. Anatomical studies of the nerve endings suggest that information from the skin must reach the central nervous system in the form of a coded space/time pattern of action potentials. Paraesthesiae, such as itching, probably result from a disturbance in the regular pattern of activity reaching the brain.

Dr. P. A. G. Monro (Cambridge) based his lecture on "The Effector Innervation of the Skin" on his thorough investigation of the effects of sympathectomy in man, which will form the subject of a monograph shortly to be published. Dr. Gordon Wright (Cambridge) spoke on "The Sensory Innervation of the Skin." After reviewing our present knowledge of the anatomy of cutaneous sensation he suggested that the reaction against von Frey's theories had perhaps gone too far. He emphasized the distinction between sensation and perception. Some functions which have been attributed to peripheral nerves may be performed centrally. Dr. R. H. Fox (Medical Research Council, Hamstead) presented the evidence for "Plasma Kinin Formation as a factor in Active Vasodilatation in Skin" and Dr. A. Scott (London) described "Acetylcholinesterase Responses in Physiological and Pathological Vasomotor Reactions." The flare of the triple response and the flare induced by intradermally injected chemicals is produced by local activation of axon reflexes. Dilatation so produced is associated with release of acetylcholinesterase from superficial nerve endings. In the absence of dilatation in eczematous skin no acetylcholinesterase is released.

Session 3: The Histochemical Investigation of the Skin

Chairman, Dr. W. N. Goldsmith (London)

Dr. A. G. Everson Pearse (London) opened the session with a lecture on "Principles of Histochemical Techniques." He gave an account of the historical development of histochemistry, of its scope and limitations and a classification of the available techniques and their underlying principles, emphasizing the steady advance of applied histochemistry. Dr. G. C. Wells (London) spoke on "Hydrolytic Enzymes in Chronic Granulomata." The results of his own investigations and of those in the literature were combined to provide a graphic demonstration of the distribution of alkaline and acid phosphatases and non-specific esterases in normal human skin and in chronic granulomata. In his lecture on "The Histochemical Investigation of Keratinisation" Dr. A. Jarrett (London) established that keratin is produced at the granular layer. Nuclei disintegrate and phospholipids can be detected; the latter perhaps supply the energy for the polymerization of the keratin precursors and the oxidation of the sulphydryl groups. Breakdown of the keratin precursors is probably brought about by the action of the phosphatases and oxidases present in the granules. Absence of these enzymes results in abnormal keratinisation as seen in psoriasis and epidermal malignancy. Dr. Kendal C. Dixon (Cambridge), speaking on "Chemical Features in Epidermal Damage," described his studies of vaccinia using the elegant oxidized tannin-azo-protein technique. Aqueous swelling was found to be a necessary prelude to intense autolysis and loss of cytoplasmic protein. The method could well be applied to the investigation of eczema.

Session 4: Bacteriology and Mycology

Chairman, Dr. R. M. Fry (Cambridge)

Dr. E. F. Gale, F.R.S. (Cambridge) opened the session with a lecture on "The Bacterial Cell" in which he described much recent work on the biochemical structure of bacteria. Dr. M. H. Gleeson-White (Cambridge) gave a detailed review of our present knowledge of the Staphylococcus, which stimulated much discussion. Dr. S. D. Elliott (Cambridge) introduced a new American film on phagocytosis. Dr. C. H. Whittle, Dr. G. A. Gresham, and Miss M. Burns (Cambridge) described recent work on Candida in man and experimental animals.

Session 5: Psychophysiological Mechanisms

Chairman, Dr. Brian Russell (London)

The opening lectures, both concerned with "Psychophysiological Mechanisms in the Psycho-

somatic Skin Affections" were given by Dr. D. Russell Davis and Dr. J. H. Cullen (Cambridge). Dr. Russell Davis emphasized the stereotyping of the pattern of reaction in attacks of migraine, peptic ulcer, asthma, and atopic dermatitis although the provoking stressor may vary. This pattern may often be established in childhood and is acquired through learning. It is possible to develop habits served by the autonomic nervous system and other systems not under voluntary control. Dr. Cullen, who shares his colleague's experimental approach, further elaborated the theme of the development of psychophysiological processes in relation to the growth of the personality with reference to recognized clinical reaction patterns.

Dr. F. A. Whitlock (Newcastle), in a "Critical Assessment of the Psychosomatic Concept Applied to Dermatology" was enabled by his experience of both specialities to evaluate and dismiss as unreliable many of the psychiatric concepts favored by some schools of dermatology. He saw no justification for the assumption that specific lesions tend to be associated with specific tensions. He suggested that central itching resulting from emotional tension might be projected to an area made vulnerable by some primary injury to produce a circular effect. Dr. B. C. G. Ackner (London) speaking on "The Relationship Between Anxiety, Alerting, and Cutaneous Vasomotor Activity" described investigations in which combined electroencephalographic and finger plethysmographic recordings were taken from emotionally disturbed and normal control subjects. Anxious subjects show relatively increased cutaneous vasoconstriction and differ significantly from control subjects in the degree of vasodilatation occurring during sleep induced by oral barbiturates. It is possible that in some cases of urticaria the disturbance occurs as a result of rebound vasodilatation during relaxation.

Session 6: Comparative Medicine

Chairman, Dr. Sydney Thomson (London)
(Held in the School of Veterinary Medicine)

P. A. Mortimer (Cambridge) presented a demonstration of "Recent Investigative Studies of Animal Skin Disease." Of particular interest was dry-feed dermatitis in piglets, a condition morphologically similar to human seborrhoeic dermatitis and certainly attributable to zinc deficiency. A. F. Jennings (Cambridge) provided numerous sections and photomicrographs illustrating "The Histopathology of Tumors of the Skin in Domestic Animals." B. M. Herbertson (Cambridge) and his colleagues showed guinea pigs afflicted with an inherited disorder of sebaceous secretion. Dr. P. Storie-Pugh (Cambridge) gave the commentary on Dr. A. C. Palmer's (Cambridge) film on "Scrapie, a Nervous Disease of Sheep Characterized by Pruritus." The intense pruritus in this transmissible disease appears to be exclusively of central origin.

Session 7: Immunology

Chairman, Dr. C. D. Calnan (London)

Introducing the session, Dr. R. R. A. Coombs (Cambridge) spoke on "Immune Phenomena in the Skin." He first gave an account of the antigens of the skin, organ, and tissue specific and individuality antigens, and discussed the antigens responsible for homograft rejection. Precise definition of terms is essential for any worthwhile consideration of autosensitisation. Ideally Witebsky's postulates should be fulfilled in order to prove the role of an auto-antibody reaction in the pathogenesis of any disease. Mr. W. E. Parish (Cambridge) in dealing with "Autosensitisation to Skin" further emphasized the need for the strictest definition of terms. He critically reviewed the clinical concept of autosensitisation and evaluated earlier investigations in the light of current immunological knowledge. He described his own work on autoantibodies to skin in the pig. Dr. P. G. H. Gell (Birmingham) in his lecture on "Mechanisms of Dermal Sensitisation" demonstrated the relevance to this specific and practical problem of new facts introduced at a still more fundamental level by speakers in other Sessions. Dr. R. A. Bruce (Cambridge) speaking on "Tuberculin Hypersensitivity" described his own work with a Purified Protein Derivative cream which raised the problems of infra-tuberculin allergy and of the effects of the persistence of antigen in the skin.

Session 8: Inflammation

Chairman, Dr. J. H. Twiston Davies (London)

Dr. W. G. Spector (London) spoke on "The Mechanisms of Inflammation," limiting himself to the endogenous mechanisms concerned in the acute inflammatory reaction. He gave examples of the latent period in the onset of such reactions; time is required for the activation of endogenous intermediary mechanisms. He discussed factors producing increased capillary permeability and the migration of leukocytes. Dr. M. Schachter (London) dealt with "Chemical Mediators in Inflammatory Processes." He described the properties of histamine, 5-hydroxy-tryptamine and the plasma kinins. All are held in the organism in inactive form but are readily releasable. No definite physiological role can yet be ascribed to them but they are likely to be released into inflammatory exudates whether or not they are primarily involved in the genesis of the reaction.

Session 9: Carcinogenesis
Chairman, Dr. F. R. Bettley (London)

Dr. L. Foulds (Pollards Wood Research Station) gave a comprehensive account of "Current Concepts of Carcinogenesis." Dr. A. Glücksmann's (Cambridge) "Comparison of Histological Features of Radiation and Chemically Induced Skin Cancer" emphasized the evident importance of the dermis in the maintenance of normal epidermal development and regeneration. Dr. D. L. Woodhouse (Birmingham) speaking on "Environmental Skin Cancer with Special Reference to Mineral Oil Carcinogenesis" discussed certain aspects of environmental skin cancer in general and then provided statistics for the use of lubricating oils in the United Kingdom and mentioned the occupations which are potentially hazardous. He then described the experimental work on mineral oil carcinogenesis in which he has been concerned. Dr. H. J. Whiteley (Cardiff) spoke on "The Hair Cycle and its Relation to Experimental Carcinogenesis." He gave a summary of earlier work on the subject, including his own. An experimental tumor analogous to the keratoacanthoma probably arises from the germinal bud of the hair follicle and is found when the resting phase of the hair is long and, therefore, where the hair is short. This is true also of keratoacanthoma and pitch warts in man. Recent experiments show that more tumors occur in short-haired than in normal or angora rabbits.

Session 10: Radiation and the Skin
Chairman, Dr. C. H. Whittle

Prof. J. S. Mitchell, F.R.S. (Cambridge) was prevented by illness from giving two lectures on the "Mechanisms of the Biological Actions of Ionizing Radiations and of Ultraviolet Light on the Skin." Mr. I. A. Silver (Cambridge) gave an account of his "Electrode Potential Studies" and the applicability of his technique to dermatological investigations aroused considerable interest. Dr. I. A. Magnus (London) was concerned with "Testing Patients for Photosensitivity." He reviewed the available techniques and described his own apparatus, making use of a Zenon arc lamp and a monochromator and some encouraging preliminary findings.

Session 11: Pharmacology
Chairman, Dr. R. H. Vickers (Oxford)

Professor W. D. M. Paton, F.R.S. (London) spoke on the "Pharmacology of Vasodilator Drugs" and Dr. P. H. Bradley (Birmingham) on "The Mode of Action of Tranquillizing Drugs." Both speakers presented much that was new and of the greatest theoretical and practical interest. Professor Mitchell was unfortunately unable to give his lecture on "The Chemotherapy of Malignant Disease."

What Dr. Rook did not convey in his summary is the "flavor" of the meeting. For example, Dr. Kendal Dixon opened his talk with a long passage from Shakespeare (recited from memory) which brought a storm of applause. Dr. P. Storie-Pugh's "clinical" demonstration of sheep with "scrapie" at the School of Veterinary Medicine was not only erudite but was presented with such a rare wit that it was one of the highlights of the meeting.

Apparently, when the course was being organized there was apprehension among some of the British dermatologists, who felt that a postgraduate course for clinicians limited to basic science would not be well received. At the end of the week-long session almost everyone agreed that the course was a unique venture in dermatologic education. Perhaps the most valuable single impression on the practicing dermatologist was an appreciation of the *scientific method* in research.

Fortunately, the Cambridge University Press will publish almost all the lectures and an abridged version of the discussion. It should prove to be a valuable resumé of selected basic science topics of interest not only to the dermatologist but also to the increasing number of investigators doing research on the skin.

Commonwealth Fellow, Oxford, 1958-1959.

IV Congressos de Dermatologia Ibero Latino-Americano e Luso-Espanhol

MAXIMILIAN E. OBERMAYER, M.D., Los Angeles

As the plural form "Congressos" implies there were really two congresses, the first in Lisbon (May 5-6), the second in Coimbra (May 8-9), separated by a beautiful all-day scenic drive to the other city.

The President of the Congress in Lisbon was Salazar Leite, the acknowledged leader of Portuguese dermatology. It was solemnly opened in the Aula of the Faculty of Medicine in the very large and modern Hospital de Santa Maria. The first morning session, with Xavier Vilanova in the chair, dealt with "microbiotic eczema of the foot." The subject was presented by Luis Pierini (Argentine) and by Salazar Leite. Individual papers followed. The afternoon session was held in the monumental new Instituto de Medicina Tropical with Luis Pierini presiding. It was devoted to leishmaniasis, with Xavier Vilanova and F. Rabello (Brasil) presenting the subject in masterly fashion and individual papers following. I was especially impressed with the quality of macroscopic and microscopic slides presented by the Brasilians.

On the second day, the morning was occupied with the clinical session at the old Hospital de Desterro where, exactly 20 years before, I had a most profitable visit with the late Sa Penella, who is known for his work on pseudoxanthoma elasticum. A great variety of interesting, well-worked-up cases was presented. No discussion was scheduled. The afternoon session again took place at the Instituto de Medicina Tropical with F. Rabello presiding. In this session the Venezuelans were most active, Kerdel Vegas presenting a film and slides illustrating the fight against leishmaniasis, and he, F. Scannone, and J. H. Chacín debating their "Memorandum concerning the Latin-American Council of Dermatology," a proposal to form a Latin-American Board of Dermatology patterned after our American Board, which had been circularized in mimeographed form prior to the session. The proposal was referred to a committee for further study.

The Second Congress, in Coimbra, M. Sampaio (Portugal) as President opened with the subject, "The Epidemiology of Syphilis." The majority of the contributors were Portuguese and Spanish. The subjects of the afternoon session were "Angiitis of the Limbs" and "Cutaneous Streptococcic Infections" with Sainz de Aja (Madrid) in the chair. Among the many contributors was our own Bob Kierland, who spoke in Spanish on "Livedo Reticularis with Angiitis." The second morning, with Espirito Santo, Professor at the University of Coimbra presiding, was given to "Free Communication." The majority of the papers dealt with fungous diseases, including the effect of griseofulvin. I was most impressed with Ayres de Sousa's (Portugal) presentation entitled "The Angio-Architecture of the Skin."

The quality of the presentations averaged that of our own best national meetings. But it is the social gatherings that give the real flavor of a congress and an understanding of the personalities. As to Portuguese hospitality, I have never experienced anything like it. It carried you from the beginning to the end on a round of banquets, one more sumptuous than the other, without let-up. If I ever needed a rest, it was after this Congress! After metropolitan Lisbon, the ancient university town of Coimbra (founded 1306), was like another world. A city, picturesquely situated on a hill above the river, dominated by its university, where students roam clad in Prince Albert coats and wide, flowing black capes and the female counterparts—a relatively late but most attractive addition to university life—in tailored black suits and the same capes. A city in which early medieval buildings, a magnificent library among them, blend harmoniously with recently erected completely up-to-date structures. The students contributed to the social functions of the Congress by presenting serenades, plays, choral singing, and folk dances.

Of the members of the Congress, I want to single out two personalities: Aldo Castellani, now living near Lisbon as private physician to the ex-king of Italy, an aged gentleman, bright as a bell; and Sainz de Aja, "the grand old man" of Spanish dermatology, Honorary President of both Con-

Submitted for publication July 1, 1959.

gresses, who reminded me of our late beloved Pusey of Chicago.

Of course, there were language difficulties. If you speak only Spanish, the Portuguese language is a barrier. And the more it is spoken in its classic form—as exemplified by Salazar Leite—the harder it is to understand! The Brasilians are easier to follow.

The Secretary, Cruz Sobral, who resembles closely the late King of Spain, was the most dynamic secretary I have ever encountered and to him, as well as to the distinguished Salazar Leite, must go the credit of a most successful congress. The outsiders, we were four: Bob Kierland, Norman Epstein of San Francisco, and E. H. Hermans of Rotterdam, were treated with every consideration we could have possibly expected—and more.

The Congress also marked the birth of a new journal *DERMATOLOGIA IBERO LATINO-AMERICANA,* Salazar Leite and Cruz Sobral, editors. No. 1, Vol. I, explains "la raison d'etre" of the new periodical, which is the organ of the COLEGIO IBERO LATINO-AMERICANO, the Association which has been sponsoring the Congresses every three years and of which the present Congress was the fourth.

CORRECTION

In an article by G. A. Grant Peterkin entitled "The Changing Pattern of Dermatology," A.M.A. ARCHIVES OF DERMATOLOGY, July, 1959, page 42/4, the author uses the word VIOFORM as if it were a common (generic) term. VIOFORM is a trademark owned by CIBA, the generic name of which is iodochlorhydroxyquin.

Clinical Notes, New Instruments and Techniques

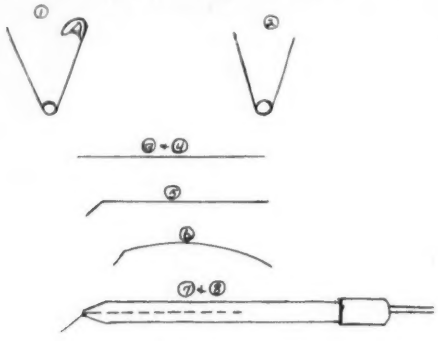
Adaptation of the Safety-Pin for Use as a Desiccating Needle

MILTON S. HARTMAN, M.D., Brooklyn

The ordinary safety-pin can be easily converted for use as a desiccating needle.

1. Open the pin.
2. Grasp the closing end with a plier and bend back and forth till it snaps off.

Submitted for publication June 2, 1959.



3. Bend the pin back on itself until it is straight.
4. Obliterate the kinks with the aid of plier.
5. Make an angle of about 45 degrees about 1.5-2 cm. from the point of pin with plier.
6. Bend shaft of pin into an arc.
7. Force shaft into electrode handle with latter in open position.
8. Tighten handle.

It is now ready for use.

Some may find it easier to do Step 3 before Step 2.

Increasing the bend in the shaft will render the needle more secure in the holder.

Necessity is indeed the mother of invention, as the above occurred to me on an occasion when I found myself without a desiccating needle to my name.

Moral: If you do not want to be annoyed with diaper-pins keep a plentiful supply of desiccating needles on hand.

Massive Fibroma Pendulum

Report of a Case

V. MEDD HENINGTON, M.D., and ANTON E. CAROE, M.D., New Orleans

Fibroma pendulum, a variant of neurofibromatosis (von Recklinghausen's disease), usually arises from the plexiform type of neurofibroma, most often on the face and neck. Few well developed cases have been recorded within the continental limits of the United States,[1-7] and Venner,[8] in a review of the classification of multiple neurofibromatosis, emphasizes the rarity of this form.

The following case report concerns a patient with fibroma pendulum which had developed to a remarkable extent before she sought medical aid.

Submitted for publication May 1, 1959.

Report of a Case

The patient, a 69-year-old white woman of French descent, was first seen at Charity Hospital of Louisiana at New Orleans on Sept. 20, 1956. Her only complaint was increasing discomfort on the left side of her face due to the continuous strain of the weight of a mass originating in this area (Figs. 1 and 2). Numerous other growths, grossly neurofibromata, on other parts of the body caused no discomfort.

According to the patient's history, which was accepted as generally reliable, her skin had been

Fig. 1.—Anterior view of patient with massive fibroma pendulum, Sept., 1956.

Fig. 2.—Lateral view, Sept., 1956.

free from any sort of lesion until she was 16 years of age. At that time, small nodules began to appear on the trunk. At the same time, the pendulous lesion now present on the left side of her body began to grow from a wide base on the face and neck below the left ear lobe. The rate of growth was slow but steady, and by the time she was 30 years of age, the mass extended almost to the umbilicus.

There was no family history of developmental defects, and no one else in the family, to her knowledge, had neurofibromatosis. Questioning elicited no evidence of consanguinity in the patient's parents.

Her own past history was remarkably free of illness. The menstrual history was normal. She had never married. In fact, she had lived a healthy and normally active life aside from the impediment of the pendulous mass. It had not occurred to her, it seemed, to seek medical relief for it during the more than half a century it had been present, although she was a woman of apparently average intelligence.

Physical examination revealed a wiry woman, who seemed about the stated age, and who had no signs of acute or chronic illness. The only skeletal defect was a slight stoop, associated with advancing age. Retinal changes were limited to mild arteriosclerosis of the central vessels. On inspection, the chest appeared mildly emphysematous, but auscultation, percussion, and roentgenologic examination revealed no abnormalities. There was no significant lymphadenopathy and no peripheral edema.

The only positive findings, in fact, were dermatologic. There were hundreds of neurofibromata, some of them up to an inch in diameter, on all parts of the trunk and extremities, as well as a few *café-au-lait* spots. Arising from a broad base on the left side of the face, neck, shoulder, and anterior-superior portion of the chest was an enormous soft, nontender, redundant, pedunculated mass which cascaded—the term is fully justified—in multiple folds almost to the symphysis pubis (Figs. 1 and 2). The surface of the mass showed fine wrinkling and deepening of pigmentation, and small, firm nodules could be palpated throughout it.

Routine laboratory examinations were within normal limits. Roentgenologic studies of the skull, chest, pelvis, and long bones revealed none of the findings of neurofibromatosis described by Holt and Wright.[9]

The patient agreed to surgery, and on Nov. 25, 1956, under endotracheal anesthesia, excision of the huge mass of skin and subcutaneous tissue was undertaken by members of the Independent Surgical Service of the New Orleans Charity Hospital. The operation was accomplished without

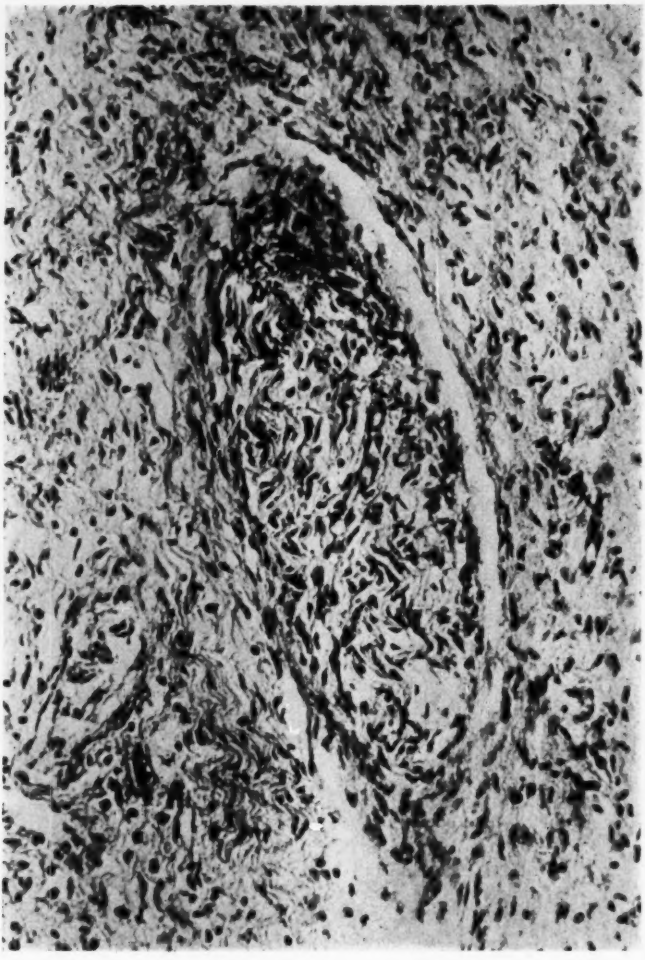


Fig. 3. — Photomicrograph (reduced 10% from mag. ×400) of typical section of excised fibroma pendulum.

difficulty, and primary closure of the incision was possible after removal of approximately 10 pounds of tissue.

The pathologist made the following report:

> Received in the fresh state were four pieces of tissue. Each is composed of very wrinkled, dirty white skin, the surface of which presents a corrugated appearance with innumerable small nodules. The two larger pieces represent folded pedunculated pouches of skin. The largest piece of tissue measures about 45 by 19 cm. Representative sections of tissue were submitted for microscopic study. Histologically, neurofibromata were seen (Fig. 3).

The patient's postoperative course was smooth, and she was discharged in good condition on Dec. 12, 1956 (Fig. 4). A few weeks later she was readmitted for plastic revision of the initial skin closure. This was successfully carried out under general anesthesia on Jan. 15, 1957. Healing was prompt and satisfactory. The patient was pleased with the results of surgery, and very much pleased to be relieved of the inconvenience and discomfort of the huge mass she had carried for so many years.

In September, 1957, the patient, who had continued to be perfectly well and without symptoms of any kind, died suddenly at her home in the country. The cause of death was assumed to be coronary thrombosis, but autopsy was not permitted.

Fig. 4.—Anterior view of patient, Dec., 1956; two and one-half weeks after excision of massive fibroma pendulum.

Comment

There are several unusual features in this case, as follows:

Careful questioning revealed no story of the stigmata of neurofibromatosis in any member of the family. This is[8] generally looked upon as a familial disease, and Frank[10] was able to trace it (Von Recklinghausen's disease) through five generations of one family as a Mendelian dominant.

Careful investigations in the hospital revealed no clinical or laboratory evidence of endocrine or metabolic dysfunction in this patient.

The most extraordinary feature of the case is that a woman of at least average intelligence should have permitted this obviously abnormal growth to reach such massive proportions and should have been willing to submit to the inconvenience and discomfort of its presence for more than half a century. Almost as remarkable is why she suddenly decided to seek medical care for it; for this, no explanation was elicited.

Summary

A case of multiple neurofibromatosis characterized by massive fibroma pendulum formation present for 53 years is described. Removal of the mass by surgery, with later plastic repair, completely relieved long-standing discomfort and inconvenience.

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Simultaneous Evolution of a Subacute Lupus Erythematosus and of Psoriasis

FREDERICK KALZ, M.D., and ZOLTAN FEKETE, M.D., Montreal

The coincidence of lupus erythematosus (L.E.) and psoriasis appears to be a rare event, and only two references have been found in the literature. Christ[1] reported one case, and Schauman[2] described three patients, in all of whom the psoriasis had been present for many years before the L.E. changes became apparent. Other instances suspected[2] turned out to be cases of lichen planus on further observation. We have not been able to find a report of a simultaneous development of these two diseases.

Report of a Case

A 37-year-old woman of English extraction was first seen in November 1957. Her past history included a tonsillectomy in 1930, a short bout of an otitis media in 1937, and a nonicteric hepatitis in 1952.

She has had no previous skin disease, but she always had a tender skin and got sunburned easily and exposure to sun was often followed by headaches. In August 1957, she suffered a severe sunburn, although she denied any excessive exposure. After the burn had subsided, a rash remained on nose, cheeks, neck, and shoulders.

She showed, when first seen, a deep-seated erythema over the nose and cheeks, and follicular plugging was noted in this area. Scattered over the shoulders were numerous drop-like infiltrated erythemas, some covered by an adherent scale. The clinical impression was that of a subacute L.E. or of an L.E.-like light eruption.

The scalp showed diffuse desquamation, and there were erythematous, desquamating, sharply bordered areas on the nape of the neck and behind the ears and similar patches over the chest and on extremities. Scratching produced the characteristic bleeding points; the clinical diagnosis of these lesions was psoriasis in a seborrheic skin. On the neck were desquamating erythemas which showed characteristics of both diseases.

She had no fever or malaise, and a general examination showed nothing abnormal. The L.E. test was negative on three occasions; the result of biopsies will be reported later.

Treatment with chloroquine (Aralen) was started with 500 mg. daily for one week and then continued with 250 mg. daily. Some improvement was noted but a sudden toxic erythema occurred after four weeks, which necessitated hospital admission. The state of her skin disorders was similar to the one described above, with an additional morbilliform eruption over the whole body, diagnosed as a drug eruption due to chloroquine.

Laboratory Examination. — The hemogram showed Hgb. 13.4 gm. %, hematocrit 41, sedimentation rate 23 minutes, total white cell count 4,000, with neutrophils 44%, band forms 4%, eosinophils 15%, monocytes 11%, and lymphocytes 15%. This was almost identical to a hemogram performed one month before, except for a marked increase in the number of eosinophils.

The L.E. phenomenon was again negative on two occasions. Plasma electrophoresis was done on three occasions, and the values remained within normal limits; the γ-globulins were high, varying between 19% and 22%. The mucoproteins were normal. A 24-hour-specimen of urine was negative for porphyrins; no albumen or cells were present, and her S.T.S. and several liver-function tests were normal. A chest x-ray and an electrocardiogram showed no abnormalities; an x-ray of the teeth gave evidence of several periapical abscesses.

She was given 20 mg. of prednisolone daily for two weeks; then dosage was gradually decreased and discontinued after four weeks. The drug eruption responded satisfactorily; the L.E. showed only a moderate improvement. After discharge, she developed several furuncles and was given a course of Staphylococcus toxoid. Her skin remained virtually unchanged during this period, apart from the development of several new psoriatic plaques on her body and extremities. During the following months she noted an increased sensitivity to sun, short exposures causing burning and redness, and eventually even exposure to light became irritating; she spent most of her time at home with drawn blinds. In December 1958, she noted increased redness and burning of the facial lesions and complained of tiredness and of occasional joint pains, and she was again admitted. Internal examination showed nothing abnormal, and repetition of the hematological, biochemical, and electrophoretic testing revealed essentially the same results as before except for an increase in the sedimentation rate to 33 mm. The L.E. phenomenon, however, had become positive, and this was substantiated by three repetitions of the test.

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From the Section of Dermatology, Department of Medicine, Royal Victoria Hospital.

Fig. 1.—Distribution of the L.E. lesions on face.

She complained of headaches, and an electroencephalogram showed minor abnormalities suggestive of a poorly localized bilateral cerebral disturbance with possibly a small area of cerebral damage in the right parietal region. These findings were not conclusive.

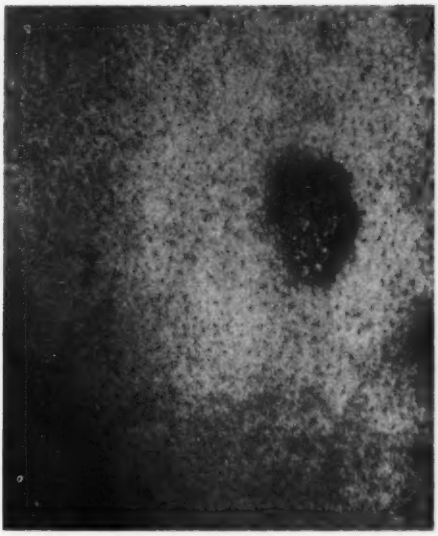

Fig. 2.—Psoriatic plaque on left side of back.

Figures 1 and 2 demonstrate the cutaneous symptoms.

Two biopsy specimens were taken; one from the neck, showing typical L.E. changes, and the second from a plaque on the left knee, showing psoriatic changes (Figs. 3 and 4).

Capillary microscopic examinations were reported by Dr. Paul Telner as follows.

1. Right hand: There are numerous regularly arranged dilated capillary loops, corresponding closely to the "cotton-ball tufts" described by

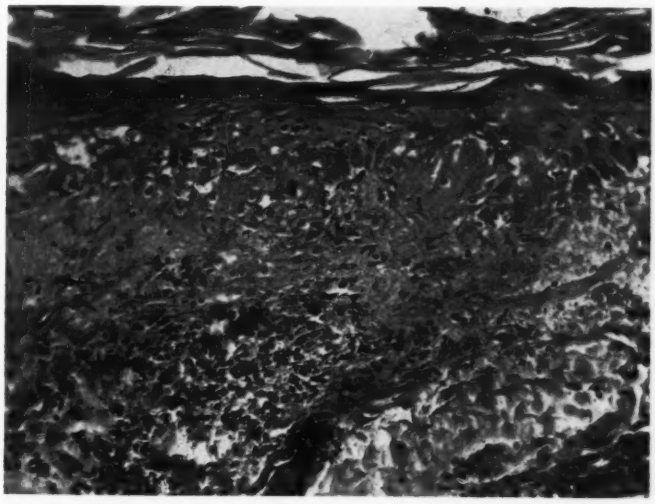

Fig. 3.—Biopsy specimen from L.E. lesion on neck. Irregular slight acanthosis, with liquefaction degeneration in basal layer, lymphocytic patchy infiltrate in upper cutis.

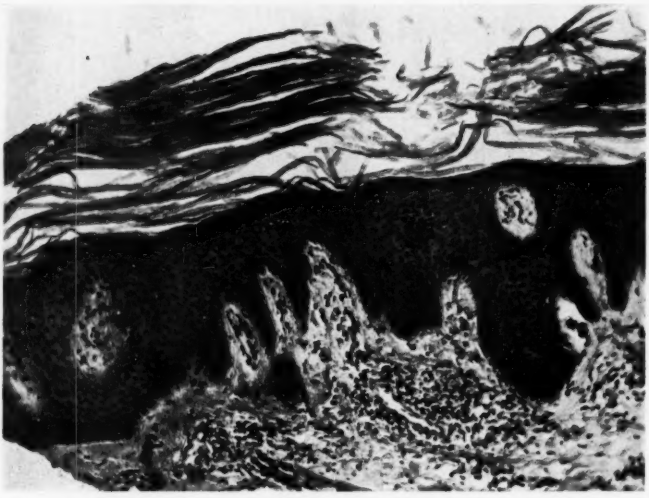

Fig. 4.—Biopsy specimen from psoriasis plaque on knee. Hyperkeratosis with patchy parakeratosis. Suprapapillary portions of rete Malpighigü are thin, capillaries in upper cutis are dilated. Moderate perivascular inflammatory infiltrate.

Gilje. This picture is characteristic for, and one may say pathognomonic, of psoriasis.

2. Face: This field shows many yellowish-brown, round structures, which represent follicular plugs. Between them is a network of irregularly dilated capillaries, with a fuzzy outline, running parallel to the skin surface. Particularly, one capillary may be seen for a considerable extent of its course, being close to the surface. There is no evidence of the typical capillary formation of the psoriatic lesion. This appearance of the capillaries is compatible with the diagnosis of an L.E. or of a polymorph light eruption (Figs. 5 and 6).

Treatment with another antimalarial drug (Triquine) was followed by severe headaches and some fever after 24 hours and was discontinued. She was then given steroid therapy using dexamethasone (Decadron). The initial dosage was

Fig. 5.—Capillary microscopic view of psoriatic plaque. Regularly arranged capillary loops, typical "cotton ball" tufts, characteristic of psoriasis.

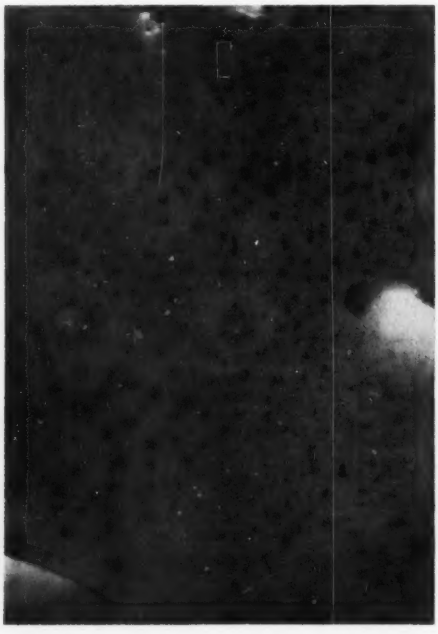

Fig. 6.—Capillary microscopic view of L.E. lesion. The round structures are follicular plugs, several irregularly dilated capillaries running parallel to the skin surface are visible.

3 mg. daily, slowly reduced to 1.5 mg. per day. Her L.E. lesions improved, while no change occurred in the psoriatic lesions.

Summary

The case of a 34-year-old woman is described who developed—following a sunburn—simultaneously a subacute lupus erythematosus (L.E.) and a psoriasis. The diagnosis of the two conditions was based on the typical clinical appearance, on a repeatedly positive L.E. test, on histopathological examinations, and on very characteristic capillary microscopic findings.

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The Transmission of Parasites Via Air Conditioners

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In substantiating a recent report by Gross,[1] I wish to comment on a similar incident which occurred last fall.

The librarian in the doctors' library at a local hospital was subjected to bites whenever she sat at her desk which was placed about six feet from a window. Frequenters of the library were also noted to scratch when spending some time in the room. The recognition of parasites as cause of the bites and pruritus became simple when numerous tiny gray-black moving particles were seen on books, tables, shelves, clothing, etc., on a warm day when the window air conditioner was in operation constantly. On top of the outside portion of the air conditioner a pigeon nest containing the parasites was found. Hence, presumptive evidence was present to implicate Dermanyssus gallinae, the chicken mite, as the cause of the dermatitis, with pigeons acting as vectors.[2]

After removal of the nest and the use of acaricides by professional exterminators, no further trouble in this respect occurred.

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Iodophors in Dermatology

A Clinical Note

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Iodine was first popularized as a surface and wound disinfectant shortly before the Civil War. Since that time its rapid destruction of viruses, fungi, and spores has been repeatedly documented in the scientific literature. Certain inherent disadvantages, however, such as staining, tissue irritation, pain on application, offensive odor, and rapid inactivation by complexing with proteins and certain drugs, led to the introduction of a host of

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From the Department of Dermatology (Dr. John R. Haserick, Chief), The Cleveland Clinic Foundation, and The Frank E. Bunts Educational Institute.

other disinfectants. None of these have, unfortunately, equaled iodine in range and rapidity of action.

Recently, several new complexes of iodine have been introduced. One, a complex of iodine with polyvinylpyrrolidone, a plasma expander, has been reported to be effective for surface disinfection and in treatment of eczematoid tinea.[1-3] Other new iodine complexes, the so-called "iodophors" have been available for the past few years. In these compounds, iodine has been complexed with various surfactants, to produce certain unique advantages.[4-7] This report discusses the clinical application of one such iodophor, Wescodyne,* containing a nonionic surfactant and iodine, as follows:

Ingredient	Percentage %
Polyethoxy polypropoxy ethanol-iodine complex	7.75
Nonylphenyl ether of polyethylene glycol-iodine complex	7.75
Hydrogen chloride	0.10
Inert material	84.40

The iodophors are used extensively in hospitals for disinfecting floors, instruments, dishes, in decontaminating attendants on isolation cases and contagion wards, and as an adjunct for preoperative scrubbing by surgeons and nurses. When properly used they are not toxic, do not irritate or stain and have no appreciable odor. Because they are a complex of iodine and a nonionic detergent, they penetrate readily into fatty materials and are not rapidly bound to proteins. They are stable under normal storage conditions and water soluble. When diluted with water for compresses or soaks the iodophor solution is initially golden-brown but becomes clear as the iodine is absorbed. Thus, the color of the solution gives a visual index of its relative germicidal potency.

Wescodyne was tested by the manufacturers on 200 human volunteers both by the Schwartz method of patch testing for hypersensitivity and by the repeated insult method of Draize and Shelanki. No significant reactions occurred and no published report of hypersensitivity to this material has been found. The bacterial spectrum and germicidal concentrations for this material have been well documented.[8]

Clinical Experience

In 1955, Wescodyne was introduced in the hospital at Sampson Air Force Base for its conventional uses on the contagious disease wards. At that time, I was dissatisfied with the conventional solutions used for treatment of infected skin diseases and gradually introduced the use of a Wescodyne solution for compresses and soaks in the concentration of one ounce of Wescodyne to one liter of water. The results were so gratifying that this material was used on virtually all skin lesions that showed evidence of primary or secondary infection with bacteria, viruses, yeast or fungi, and various dilutions up to and including the concentrated form were tested. In the past four years this has been used on several hundreds of patients without development of true hypersensitivity and only rare instances of irritation, none of which necessitated discontinuation of the medication.

The following tabulation summarizes the clinical impression of Wescodyne:

- Excellent Results
  - Kaposi's varicelliform eruption
  - Secondarily impetiginized dermatoses
  - Mixed intertriginous infections
  - Infectious eczematoid dermatitis
  - Molluscum contagiosum
- Very Good Results
  - Herpes zoster
  - Folliculitis
  - Hidroadenitis suppurativa
  - Dermatophytosis
  - Monilial paronychias, Onychomycosis, Flat or juvenile warts } Applied full strength
- Satisfactory Results
  - Pustular bacterid
  - Dermatitis repens
  - Furunculosis

After considerable experimentation with the various dilutions of Wescodyne, the original one-ounce-to-one-liter dilution has been most satisfactory with the exception of thickened indolent lesions, where application without dilution is employed. The nursing staff has been favorably impressed by the ease with which this material is handled and its lack of permanent staining. The patient is much impressed by seeing the color fade as he carries out the prescribed therapy, and this psychologic aspect undoubtedly contributes to some of the excellent results and has perhaps induced the patient to be more persevering in therapy.

The most striking results with this iodophor compound have been in treatment of some of the viral diseases. Three patients with Kaposi's varicelliform eruption admitted to the hospital with extensive cutaneous involvement, malaise, and high fever, within 12 hours of continuous wet compressing with Wescodyne all exhibited striking improvement of the skin, normal temperature, and lessened malaise. Since the compresses practically covered the entire body surface, and we were concerned with possible absorption of iodine, they were discontinued at the end of 24 hours without recurrence of the symptoms.

* West Chemical Company, 4216 West St., Long Island City, N.Y.

Although the flat warts of children and young adults have been treated with various methods, including hypnosis and suggestion, the results of painting full-strength Wescodyne on these lesions several times daily have been most gratifying. In no instance was there any evidence of tissue irritation. While it is possible that suggestion and/or natural immune factors may have caused resolution of these verrucae, the penetration of sebum and keratin by iodophors would suggest that viricidal amounts of iodine may reach the base of these thin lesions. This material has also been helpful in the treatment of plantar warts after maceration or irritation by chemical therapy and considerably shortens the cutaneous manifestations of herpes zoster.

Summary

A new type of detergent iodine complex of the "iodophor" group has been used in the treatment of primarily or secondarily infected skin diseases for a four-year period and has been found to be effective over a wide range of infectious agents. Its relative freedom from toxicity, irritation, staining, and odor have made it less objectionable than its older counterparts.

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Wart Relapses at the Edges of Therapeutic Cantharidin Blisters

GEORGE H. FINDLAY, M.D., Pretoria, South Africa

When Epstein and Kligman[1] described the treatment of warts with cantharidin they mentioned as an unusual complication a "rapid growth of the wart to occupy the entire blister site. A 'doughnut' formed, with apparent central clearing and a peripheral ring of exuberant new wart."

The accompanying illustration shows a well marked example of this phenomenon. It comes from a 17-year-old girl whose forearm was treated for about 10 small warts with Kligman's 0.7 per cent cantharidin varnish. Large blisters, one with pseudopodia, resulted. These were not drained but were treated by occlusion with a fatty gauze dressing. The dry blisters peeled and six weeks later the picture of polycyclic and circinate patterns of wart papules was seen, showing in one

Gyrate, circinate, and iris arrangement of warts in the pattern of preceding cantharidin blistering.

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From the Section of Dermatology, University of Pretoria (Head: Dr. G. H. Findlay).

lesion an iris arrangement through persistence in the center of the original wart (Figure).

This phenomenon seemed to represent an intraepidermal autoinoculation of the wart virus through the blister cavity, with development of the wart in the site of the outer angle of the blister cavity within a few weeks. Inoculation by other means usually takes about four times as long to produce a growth.[2]

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Erythema Gyratum Repens

Report of a Case

M. J. PURDY, M.B., Ch.B.(N.Z.), M.R.C.P.E., Hamilton, New Zealand

In October, 1952, Dr. John A Gammel reported a case of a woman with a bizarre skin eruption apparently related to cancer of the breast. He labeled it "erythema gyratum repens." No subsequent publication in English of a case closely similar seems to have been made.

The salient features of Gammel's case were as follows:

1. Woman aged 55 years.
2. Eruption present for nine months.
3. Began on the anterior neck and adjacent part of the chest.
4. Face and neck reddened and scaly; edema of lids.
5. Trunk and limbs covered by erythema with a pattern like a knotty cypress board (excellent photographs show this well) and with a collarette type of desquamation; the erythema moved from day to day and parts would clear while others flared.
6. Poorly differentiated adenocarcinoma of the breast discovered.
7. Rapid clearing of the rash following removal of the growth.
8. Pruritus of the initial area involved and of the scalp mentioned but was not stressed with regard to the main eruption.

He mentioned briefly the chief features of the other rare erythemata and concluded that erythema gyratum repens was sufficiently distinctive in character to warrant the new title.

Erythema Gyratum Repens.

Report of a Case

The patient was a woman 45 years of age and came with an eruption which had been present for three months. The lesions were strikingly bizarre and brought to mind the photograph in Gammel's article. The similarity between that photograph and the photograph of the present patient is, as may be seen, strong. A figurate eruption covered the trunk and limbs. The lesions were slightly raised and erythematous and collarette type of desquamation was evident. The borders of the lesions advanced slowly from day to day, and there was clearing behind. However, new lesions would soon begin in the recently healed areas. The pattern brought to mind that of timber graining, as noted by Gammel. The face and neck were florid, and there was slight edema of the eyelids. The eruption had begun over the right breast. Itching was severe and there was general malaise.

A general clinical examination revealed a small hard and painless lump in the right breast. There were no palpably involved lymph nodes, and other organs and blood and urine were apparently normal.

After a radical mastectomy the itching was gone in twenty-four hours; the erythema had gone by the third day and was succeeded by profuse desquamation which lasted for a week, after which the skin appeared normal.

The breast removed was shown histologically to be involved in a diffuse duct carcinomatosis.

Summary

A further case of erythema gyratum repens is described. Its similarity to that described by Gammel is so striking as to support his claim that the eruption is distinctive. Because of the findings in his case, special care was had in examining the breasts and led to the discovery of a tumor of which the patient had been unaware.

Nolan Building, Hood St.

Addendum

Since the above article was written a further case of erythema gyratum repens has been published (*Proc. Roy. Soc. Med.* 52:367 [May] 1959) by J. Schneeweiss, M. B. (for S. C. Gold, M.D., F.R.C.P.).

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Correspondence

STEROID OINTMENTS IN DERMATITIS

To the Editor:—In a previous paper, Scott and I reported on experiments which suggested that the course of experimentally induced skin inflammation was not altered by external application of hydrocortisone, if applied after the inflammatory stimulus was set.[1]

Nevertheless, and in apparent contradiction to these findings, we have been impressed with the satisfactory effect of hydrocortisone creams in a number of inflammatory skin disorders, including contact dermatitis.[2] A great number of reports on the efficacy of steroid ointments in contact dermatitis has been published from all over the world, and it appears that these preparations are used routinely by the majority of dermatologists in their daily practice.

Kligman, in his excellent article on poison ivy dermatitis[3] digressed to castigate the indiscriminate use of steroid ointments in various skin diseases and singled out, as an example, our clinical report on the effect of hydrocortisone creams on contact dermatitis. To support his views, he made reference to his experiments with a variety of contact allergens given at threshold concentration, in which steroid ointments failed to alter the course of the reactions. While he did not give details of his technique used and of the number of patients tested, I do not doubt the accuracy of his experiments. Nevertheless, the question might be raised whether such model experiments are comparable to the actual clinical state of a patient with contact dermatitis. Allergic sensitization, or any other inflammatory stimulus, may initiate a process of inflammation; there is evidence that externally applied steroids do not inhibit this initial phase of the reaction; the further course may be short lived and self limited and external applications may not alter this course. The clinical picture of a contact dermatitis often differs from his model, it may persist long after the contact allergen has been removed and the dynamics of such a case appear to be more complicated. Patients seen in outdoor departments tend to fall in this category. On the other hand, I realized that we had offered no proof for the validity of our clinical observations, and I felt at the time that an answer would be of polemic character and would serve no purpose.

Recently, however, we have studied the effect of three new steroid compounds as compared to an empty control ointment, and one containing hydrocortisone, using the double-blind paired comparison method. Patients with atopic and with contact dermatitis were chosen and over 700 test sites were compared, the majority cases of contact dermatitis. All but two patients were able to spot correctly the empty control ointment within a few days, and a good many interrupted the experiment, stating, "This ointment is no good."

These findings corroborated our clinical impressions but were in contrast to Kligman's as well as to our own experimental results. They also did not confirm a recent report[4] claiming little differences in the effect of steroid lotions and empty controls in a number of dermatoses, which may be due to a different selection of cases.

Pondering upon these discrepancies we realized the difficulties in assessing the relevancy of the selection of cases, of the timing of applications, of the stage of the inflammatory changes, the location of the disorders and of many other variable factors. One of these is the motivation of the investigator and it seemed to us that unconscious bias is difficult to eliminate, even when using appropriate controls. Innumerable erroneous statements have appeared in the literature and the source of the error has often been prejudice. The prejudiced observer may appear in two forms—the uncritical enthusiast and the denigrating debunker. The former is too well known to be discussed, the latter may best be described by quoting Samuel Johnson: "Truth, Sir, is a cow, which will yield sceptics no more milk; so they have gone to milk the bull."

Of course, I do not mean to imply that I count myself into the former and Dr. Kligman into the latter category, but it is axiomatic that truth and proof must not be confounded.

An experiment may present proof within the framework of its setting; it may not reveal truth. On the other hand, an observation may remain unproven for some time and may eventually found to be true.

Such reflections tend to leave one with a sense of humility. Colton said: "The greatest friend of truth is time, the greatest enemy prejudice and her constant companion is humility."

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DR. KLIGMAN'S ANSWER

To the Editor:—Dr. Kalz has a winning letter. It is an artistic creation. The very thought of opposing it seems sinful. His argument is in the classical tradition set up by Gladstone; it is balanced, in good taste, sober, and eminently fair. I certainly admire Kalz's literary craftsmanship. Furthermore, I could wish to have written it myself for his thoughts have great merit and are gently delivered. As is well known, I prefer rapiers to the back of the gloved hand.

We are, however, not involved in a literary contest. Words and thoughts, no matter how beautifully assembled, will not decide the real issue. Scholarship by no means guarantees access to the truth. I suspect that Dr. Kalz and I have the same philosophy of research. We differ only in regard to what we think we have observed. I cannot verify his observations. I do not think this is due to the Philadelphia weather or the obvious defects in my upbringing. I am annoyed when I cannot reproduce what others have done. By this standard anyone engaged in dermatologic research has a plethora of opportunities for annoyance. I should certainly urge anyone interested in a full time debunking career (either of the denigrating or deviling variety) to enter dermatology. We do not suffer from *lack* of knowledge, as our critics never tire of telling us, but from *bad* knowledge.

To get to the heart of the matter, Scott and Kalz claim that prior application of topical steroids suppresses the inflammation produced by mustard oil, nitric acid, and ultraviolet rays. For two years at least, I have been trying to confirm these findings and similar ones of others without success. Indeed, we have been unable to devise an acute experimental inflammation which could be moderated by topical steroids. (Witkowski and Kligman: J.I.D. 32:481, 1959). We need not enlarge the argument to include the disparity between the success clinicians routinely achieve with topical steroids in allergic contact dermatitis and my own impotence in treating the same disease produced experimentally. To settle our disagreement, it is only necessary to do a very simple thing which does not require the engagement of the higher centers, as in philosophic controversy. One need only show that prior rubbing of steroids into the skin will retard or diminish the dermatitis produced in that skin by nitric acid, mustard oil, or ultraviolet rays. The same negative result is obtained regardless of when the steroid is applied, how often, and whatever the mode of application.

As a matter of fact, it is not only in the laboratory that the effectiveness of topical steroids has been exaggerated. I venture to say that more than 90% of the users of such agents would do just as well with the base. The undoubted remedial effects of topical steroids are limited to a small group of chronic dermatitides. This is perhaps a good thing for if topical steroids were anywhere near as effective as their prodigious use would seem to indicate, dermatologists might be rendered obsolete. I even have a good word for drug houses. I should counsel against dropping the price of topical steroids for the surest way for practitioners to discover the matching therapeutic qualities of boric acid ointment and topical steroids in most skin conditions would be when the price became the same. Finally, one may not be able to get milk from a bull, but it is good exercise.

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Philadelphia 4

HISTORY OF ACNE

To the Editor:—In the July issue of A.M.A. ARCHIVES OF DERMATOLOGY there is a short note by Dr. Weiner on a case for diagnosis by Chaucer. Chaucer's passage has been commented on before. Dr. R. N. R. Grant, in an interesting article on the "History of Acne," which appeared in *Proceedings of the Royal Society of Medicine* 44:647-652 (Aug.) 1951 (Section of the History of Medicine, pages 19-24), quotes the same passage and suggests a diagnosis of rosacea.

ADOLPH ROSTENBERG JR., M.D.

AUDIO-TAPES FOR DERMATOLOGY

To the Editor:—I have been very much impressed with the use of the audio-tapes available to the various specialties. The men who have been using them have been most enthusiastic and not only do they cover the salient literature but also present highlights of the various national and international meetings, round tables, and symposiums.

Unfortunately, Dermatology is one of the few specialties for which this service is not available. I cannot help but feel that all the members in our specialty group would benefit by and appreciate the efforts of our leaders to make this service available.

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COST OF GRISEOFULVIN

To the Editor:—I was detailed today by telephone, telegram, mailing, and in person on the United States release of griseofulvin. According to two different detail men, this is to cost the patient 40 and 60 cents a tablet. (My previous experience is that drugs cost more than detail men say, but leave us accept this figure.) One brochure recommends eight tablets daily, the other four. By wiggling my slide rule, for a "normal" course of therapy over a six-month period, the patient can spend $864.00 for 1,440 tablets of griseofulvin!

In the May, 1959, *Brit. J. Dermat.*, griseofulvin is advertised at a retail price of $8.40 (60 shillings) per hundred, or $78.40 (560 shillings) per thousand. This would bring the cost of 1,440 tablets to a total of $113.00 in Great Britain. Since the round-trip airline fare from New York to London is only $462.00, a patient who plans to take griseofulvin in full therapeutic dosage can save by making a week end trip to London to get his prescription filled.

One California dermatologist has already had friends sending him griseofulvin from Malaya for 9 cents a tablet, including air mail postage. Until customs have been educated to cut off this source of supply, there may be a future for griseofulvin smugglers. I can see the eagle eyed customs inspector finding the white powder in the false bottom of the suitcase, and saying triumphantly, "Ahah, griseofulvin," while the culprit begs for mercy on the grounds that it is only heroin!

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Obituaries

ERICH HOFFMANN, M.D.

1868—1959

Erich Hoffmann, professor emeritus of the University of Bonn, Germany, died May 8, 1959, shortly after his 91st birthday. He was the nestor of the German dermatological school and one of the leaders for many years. Dermatological societies all over the world honored his merits by bestowing honorary memberships upon him. Up to the last years he took an active part in the development of our specialty and continued to publish important papers, reflecting his rich clinical and scientific experiences.

He was born April 25, 1868, in Witnitz, a small town in Pomerany, Germany, the son of a minister. He obtained his medical education, like so many important medical men, in the Military Medical Academy of Berlin. After a relatively short military tour, he was assigned to the University Skin Clinic in Berlin, which was under the direction of Edmund Lesser. In a short time he acquired a prominent position in clinical work and in research. In 1909, he became director of the Dermatological Policlinic at the University of Halle and, in 1910, director of the University Skin Clinic in Bonn and one of the first full professors of dermatology, a position he retained until his forced retirement in 1934.

In all these places Hoffmann was eminently active as a clinician, researcher, organizer, and teacher, and many of his pupils held important professorships all over the world. For many years he was editor of the *Dermatologische Zeitschrift* and co-editor of the German *Archives of Dermatology and Syphilis*. His work encompasses all parts of our specialty, and some of the clinical entities bear his name, such as perifolliculitis capitis abcedens and suffodiens, and the verrucosis generalisata. He was the first to recognize the importance of the skin as a protective and curative organ, a property he called "eisophylaxie." But his major interest lay in syphilis research, and his name immortally tied to one of the great medical achievements, the finding of the Spirochaeta pallida. In his memoirs he gives a very dramatic account of this discovery. He describes how Schaudinn, during joint examination of preparations of a syphilitic papule, suddenly saw one very fine pale spirochete, which was entirely different from the numerous coarse spirochetes, and immediately suspected in it the long searched for syphilis microbe, and he describes how much hard work was necessary to make sure that the enigma of the syphilis etiology was finally resolved. Together with a great number of co-workers and pupils from all over the world, Hoffmann took a prominent part in experimental and clinical syphilis research which finally led to the great therapeutic achievements of Ehrlich.

As a man, he always stated frankly his convictions. He never deterred from any remark he believed justified. And so he fought Naziism and anything that stood for it in his beloved Germany. He openly and fearlessly spoke out against Hitlerism publicly in his lectures and in private conversations. His indignant and disdainful words brought about his sudden forced retirement in 1934.

During his retirement he visited foreign countries to give lectures and revive old friendships. In his memoirs he speaks of the happy days he and his wife spent with old friends in the U.S.A., among them Alderson, Howard Fox, Pusey, Udo Wile, and many others. These two volumes of his memoirs are a rich source for anyone interested in the development of medicine in the first decades of our century.

To the last he remained extremely active in the happy companionship of his wife, Toni, and we all, his numerous visiting friends, to whom they made their charming house a home, will remain attached to them in veneration and friendship.

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The New, Newer, and Newest

California is said to attract the incredible number of 1,500 new residents a day. This makes understandable the rapid growth of the Pacific Dermatological Association, made up largely of Californians, a few of them native born and home grown, and which now has over 300 members. More than 200 members attended the eleventh annual meeting in Carmel-by-the Sea, Sept. 9, an unusually high attendance for a sectional meeting. There was much to see, much to learn, and much to enjoy, but especially striking was the informality and spirit of friendliness that prevailed.

"Fletch" Hall's Presidential Address was titled *Ars Longa, Vita Brevis.* It struck just the right tone, a sincere, thoughtful, appropriate address for the occasion—essentially a plea that we be more than skilled technicians, that we should know better the behavioral sciences. The younger men listened with interest, the older, nodded in approval.

He was followed by Joseph Peterson of Stanford, the winner of the first Nelson Paul Anderson Essay Contest, who presented "An Evaluation of Rubefacients."

Other guest speakers were Henry Michelson and Walter Lever and, from abroad, Gustav Asboe-Hansen and Alfred Marchionini. To my regret Marchionini's arrival was unavoidably delayed, and my departure was unavoidably hurried so that there was no opportunity to hear his talk, but I did hear the others, and it was good worthwhile listening indeed—Michelson on the subjects of Leukoplakia and Lupus Erythematosus, Asboe-Hansen on Pathogenesis of Cutaneous Myxedema and Modern Mesenchymology and Lever on the Xanthomatoses.

Lever also conducted the now famous C.D.A. Histopathology Round Table discussion. Where else but at this Society can you hear such give and take as, Lever: "I do not believe there is such an entity." Member: "It's in your book." Lever: "It shall be omitted in the next edition." It was worth the trip to the coast to learn of the golf-ball-sized cutaneous tumor which histologically suggested the diagnosis of ectopic brain tumor. And it was comforting to some of the older clinicians to observe that histopathologists have uncertain moments also.

The meeting was unusual in that the program did not specifically include the subjects of griseofulvin, cortico-steroids, or antibiotics, though these were all considered in the panel on therapy conducted by Arthur Woodburne.

The excellent program plus the thoughtfulness with which the social activities are planned is in large part responsible for the enthusiastic attendance at these meetings. Before getting down to serious business, there was an outdoor pool-side "get-together" under "unusual" weather conditions to which a recently transplanted easterner had the temerity to bring a raincoat but not to wear it. But the natives oblivious to the "moist" air soon found their "Scotch-on-the-rocks changed to Scotch mist. And those who failed to get together that night could not avoid it on another night at Monterey on the occasion of a buffet supper and show which defies description—the members and guests will long remember it!

The new officers are:

Louie Winer ..... President
John Ambler ..... Vice-President
Edward Ringrose ..... Secretary

Next year they will meet in Victoria, B. C. It should be an interesting meeting for the organization is young, spirited, energetic, sprawling, friendly, and creative—and wise too for they elected me an Honorary Member.

H. R.

Society Transactions

BRONX DERMATOLOGICAL SOCIETY

Arthur B. Hyman, M.D., President
Henry Silver, M.D., Secretary

February 19, 1959

Diagnosis: Herpes Simplex of a Hand (Due to Contact with Sibling who Had Herpetic Gingivostomatitis?). Presented by DR. ALEXANDER A. FISHER.

A baby of 7 months has had an eruption on a hand for 10 days. The eruption began with a small blister on a finger. Since then, crops of umbilicated vesicles, pustules, and small hemorrhagic bullae have appeared in the region. At the moment there are numerous vesicles and pustules, some of which are umbilicated. There is an enlarged lymph node in the left axilla. The temperature is not elevated, and there are no systemic symptoms.

According to the pediatrician, the patient's older brother had herpetic gingivostomatitis three weeks ago. The patient not only was in intimate contact with her brother, but was seen by the mother to place the involved hand in her brother's mouth.

The patient has already been treated with various antibiotic and antifungal ointments. Suggestions for further therapy are invited.

Discussion

DR. HARRY SHATIN: We are dealing in this child with primary inoculation herpes simplex. A clinically symptomatic phase of primary infection with herpes simplex virus is seen in less than 1% of the cases, and then it is usually in the form of an acute herpetic gingivostomatitis. The two most dangerous forms of primary herpetic infection are (1) the meningoencephalitic, which is indistinguishable clinically from other types of viral encephalitis, and (2) the visceral, which may be associated with a viremia which may be, and usually is, fatal.

In the present case, the disease process has already been present for 10 days, and is approaching the end of its course. I would treat symptomatically, and give antibiotics for any secondary bacterial infection that might develop. I would not use corticosteroids because there is risk of spread of the viral infection, and possible transformation of a condition, which is now essentially localized and benign, into the highly dangerous encephalomyelitis or viremia.

DR. IRVING D. EHRENFELD: Consideration ought to be given to the possibility of a virus other than that of herpes simplex causing this lesion. For instance, the Coxsackie virus has been shown to cause herpangina, a condition not unlike herpetic stomatitis.

If this virus could be identified, either in the patient's cutaneous lesion or in the brother's mucous membrane lesions, the whole matter would be worth wider publicity.

DR. MAX BRAITMAN: The use of aureomycin in herpes simplex involvement of the mouth has been reported by Baer and Miller, Fisher and Leider, and Distelheim and Sulzberger. All of these workers observed excellent results from aureomycin. In 1950, I reported a case of resistant, persistent, recurrent herpes simplex of the mouth of many years' duration, which responded dramatically and remained well after the use of aureomycin by mouth.

DR. FRANCES PASCHER: I don't think fancy treatment is indicated in this case. Except for the application of spirits of camphor in the initial stage of herpes simplex, I know of no therapeutic approach that is worthwhile beyond simple topical agents that are physically protective.

DR. SAMUEL B. FRANK: With regard to treatment, I have not been impressed with the effectiveness of any agent for the treatment of an individual attack of herpes simplex. I feel the real problem here is not the care of the attack, but that recurrences may follow. These would most likely be at the site of the present eruption—on the hand—and might be quite disabling.

The management of recurrent herpes has been a subject that has interested me for several years. I reported on the use of a formalin-killed herpes vaccine prepared from infected rabbit brain in 1938 (Frank, S. B.: Formolized Herpes Virus Therapy and the Neutralizing Substance in Herpes Simplex, J. Invest. Dermat. 1:267-282 (Aug.) 1938.) Treatment with this type of vaccine led to some serologic observations of interest, but its usefulness as an office procedure was found to be questionable. It also should be pointed out that the

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form of vaccine that is processed from infected brain tissue is now discouraged, because odd neurological consequences may occur.

Diagnosis: Angiokeratoma Scroti with Associated Lesions in the Mouth. Presented by Dr. Irving D. Ehrenfeld.

A 62-year-old white man developed some nodular lesions in the left side of the mouth about four years ago. About four months ago, he developed similar lesions on the right side. Scrotal lesions were noted about nine months ago. Except for some pruritus of the scrotum, and an awareness of the mouth lesions, there has been no discomfort. None of the lesions has ever bled.

Examination reveals no abnormal findings except lesions in the mouth and on the scrotum. On the scrotum there are many scattered, deep red units, 1-3 mm. in diameter, which appear like typical lesions of angiokeratoma scroti (Fordyce). In the mouth are two groups of slightly elevated purplish lesions, each situated on the buccal mucosa, just inside the right and left commissures. These empty slowly on pressure.

A mouth lesion from the group on the left was excised for histological examination. Dr. Charles Sims reported as follows: "The epidermis is moderately and irregularly acanthotic. The corium presents dilated vessels, many filled with red cells. Some edema of the collagen is present. Diagnosis: angiokeratoma."

Discussion

Dr. Frederick Reiss: It is my impression that the lesion of the lip is a hemangioma, and not an angiokeratoma. I found no evidence of hyperkeratosis on the section of the lesion of the lip, only dilated blood vessels in the upper cutis.

Dr. Arthur B. Hyman: The scrotal lesions described by Fordyce are not always keratotic, so that the term angiokeratoma is a misnomer. Similar lesions are not rare on the labia of the vulva, particularly the labia majora. Sometimes a patient's attention is drawn to their presence by sudden bleeding, which is likely to occur while in a bathtub. They respond easily and well to electrodesiccation, but new lesions commonly develop.

Dr. David Bloom: The eruption of the scrotum presents a classical example of angiokeratoma scroti as described and photographed by Sutton (Sutton and Sutton: "Diseases of the Skin," Ed. 10, St. Louis, The C. V. Mosby Company, 1939, p. 668). Hyperkeratosis is not the essential part of this dermatosis, and is frequently absent. If the lesion of the lower lip in this patient presents the typical histologic picture of angiokeratoma, it is, I believe, the first case in this location ever reported. To me, the lesions of the lip appear as simple angiomata.

Dr. Morris Leider: I wonder whether too much is not being made of the surface characteristics of angiomata. May it not be that what happens in the stratum corneum over an angioma depends merely on the level at which blood vessel replication exists, and the site of the angioma? That is to say, if hamartomatous angiomatosis is very high in the corium, especially so high that the epidermis is distorted and thinned, as it is in classical angiokeratomata, then stratum corneum hypertrophy occurs as a part of the protective phenomenon against rupture of the fine vessels. This is more likely to happen on the hands, where hyperkeratosis occurs readily. On protected parts of the body like the scrotum it is less likely, unless friction attends the situation. In mucous membrane sites, hyperkeratosis is also less likely, because of maceration, and inherent smaller ability of such epithelium to hypertrophy. Other angiomata, like nevus flammeus, spider nevus, ruby spots, and strawberry and cavernous angiomata, are all more deeply situated, superficial as they may appear to the eye, and are more completely enveloped with corium. Therefore they hardly invite or incite epidermal hypertrophy.

Dr. Irving D. Ehrenfeld: This patient has been presented because he presents typical angiokeratomata of the scrotum (Fordyce), and angiomatous lesions on the labial and buccal mucosa, a combination which I have never seen or read of before. Typically, mouth lesions may be found in angiokeratoma corporis diffusum universale of Fabry, and occasionally in angiokeratoma of Mibelli. The association of mouth and scrotal lesions of the Fordyce type noted here is worth recording for possible evaluation in the future, if other instances such as this are observed.

Diagnosis: Idiopathic Atrophoderma of Pasini and Pierini. Presented by Dr. John Garb.

A woman, now in her late twenties, was first presented in her teens at the New York Academy of Medicine by the Late Dr. Fred Wise, with the diagnosis "Pigmentation and Atrophy Preceding Morphea" (*Arch. Dermat. & Syph.* 57:525, 1948). The description of the lesions at that time was as follows: ". . . . On the . . . back, abdomen and . . . thighs [there] are dark brownish to blackish irregular spots [that] vary in size and [measure] from 1 to 7 cm. [in diameter]. They are not infiltrated. . . . They do not alter on diascopic pressure . . . The lesions [have] never regressed [and] there are no subjective symptoms." Histologic findings at that time were reported by Dr. Charles Sims as "insufficient for a precise diagnosis."

The patient is re-presented at this time with the same clinical appearance, and under the title above. Histologic examination now, again interpreted by Dr. Sims, is as vague as before.

Fig. 1.—Clinical aspect of the atrophoderma of Pasini and Pierini. The arabic numerals denote the following: (1) is abutting normal skin, (2) is a depressed atrophic lesion, and (3) is the site of biopsy which shows a linear scar and 3 punctate suture-point scars, all of which are sclerotic and raised.

Discussion

DR. DAVID BLOOM: I have seen a number of such cases presented at various society meetings in recent days, and I believe that they belong to the group of morphea, with a conspicuous feature of atrophy. I have seen such cases associated with more typical lesions of morphea. Although no increase in consistency of the skin is present, this is also true of many cases of indubitable morphea. The grayish-brown pigmentation is also seen, not infrequently, in circumscribed scleroderma. The patient states that the lesions on the thighs were white before becoming pigmented.

DR. HENRY SILVER: Though described some 35 years ago by Pasini, very few cases of this type have been shown or discussed at meetings. In South America, however, Pierini and his associates collected through the years the largest number of cases, and these observers tried to establish a separate entity under the title of idiopathic, progressive atrophoderma.

The clinical picture is that of localized atrophic patches, which give the impression of morphea in an atrophic stage. The lesion is soft, smooth, and depressed, and the border well-demarcated by a decided slope. There is no erythema to denote inflammation, no lilac ring, nor firmness as seen in scleroderma, and no sense of herniation as appreciable in macular atrophy. The histology shows a constant feature: atrophy of the mid and deep cutis. There are no elastic or connective tissue changes.

A similar case, presented before this Society a few years ago by the late Charles Rein, evoked a discussion which favored the diagnosis of cardboard scleroderma, or morphea. To my mind, the affection is a distinctive type of macular atrophy of the midcutis with an important histologic feature. Not much is gained by attaching eponyms, nor should such archaic terms as "idiopathic" and "progressive" be retained. It is a matter of another distinctive macular atrophy, cause unknown.

DR. FRANCES PASCHER: I believe this patient presents a distinctive clinical picture. Having seen a case of idiopathic atrophoderma of Pasini and Pierini presented by Dr. Canizares at the staff meeting of the New York Skin and Cancer Unit last year, it was possible for us to identify the case under discussion. As a matter of fact, on the basis of an earlier report in the transactions (*Arch. Dermat. & Syph.* 57:525, 1948), Dr. Canizares included this patient as a probable example in his paper (Canizares, O.; Sachs, P. M.; Jaimovich, L., and Torres, V. M.: Idiopathic Atrophoderma of Pasini and Pierini, A.M.A. ARCH. DERMAT. 77:42, 1958, ref. 23).

I feel it is helpful to be able to separate one atrophoderma from another, even in a tenuous manner, and therefore the concept of Pasini and Pierini, in my opinion, should be retained. The cases sorted out by them appear to be different from the anetodermas of Schweninger-Buzzi and Jadassohn. Moreover, this atrophy, in my opinion, is primary, and not secondary to scleroderma. Because of atrophy attended by hyperpigmentation, the "normal" skin about the lesions appears elevated and paler, thus giving an over-all illusion of scleroderma. Two biopsies in this case failed to disclose evidence of scleroderma.

Dr. Garb's presentation is most instructive, also, because we have been given an opportunity to follow the course, and to learn something about the prognosis in this form of atrophoderma. There apparently has been no progression in 10 years.

DR. JOHN GARB: The original presentation of this patient by Dr. Wise provoked a lot of discussion. Dr. Sulzberger favored morphea as the most appropriate diagnosis. Dr. Wilbert Sachs said the picture was part atrophic scleroderma, and part poikilodermatomyositis vasculare atrophicans. Dr. Isidore Rosen stated that his impression was of the end result of a preceding eruption of toxic origin. Dr. Peck did not think the changes were those of localized scleroderma. My own diagnosis is morphea (with unusual features). But I believe the eponyms of Pasini and Pierini are deserved simply as a distinguishing mark of the type.

Diagnosis: Leprosy (Tuberculoid Type). Presented by DR. ANDREW G. FRANKS and DR. RICHARD K. SCHER (by invitation).

A 28-year-old Negro woman was born in British Guiana, and lived there most of her life. She came to this country six months ago. About seven years ago, she noted a lesion on her left leg, and since that time more process has developed on her arms, legs, and face. The patient states that she does not have pain sensation in certain of these areas, and, on this account, has burned herself unwittingly several times.

Upon examination, the lesions of the face appear sarcoid-like. The ulnar nerves are thickened. Prick-pain and heat-pain are absent in many involved sites.

Laboratory study to date has revealed a tuberculin-type reaction (1+ in 48 hours) to lepromin, and a histopathologic picture that is compatible with tuberculoid leprosy. On the negative side, no acid-fast rods were identified in nasal scrapings, and several histamine tests were "negative."

Discussion

DR. FREDERICK REISS: The clinical features of this case are characteristic of the serpiginous form of tuberculoid leprosy. There are, however, two findings in this case which require explanation.

The presence of a flare in the histamine test performed upon an anesthetic area is not true to form. If this case is indeed one of maculoanesthetic leprosy, then one must assume that the axone reflex, upon which the histamine flare depends, is not yet ablated here.

The 24-48 hour reaction observed from the lepromin used, which, I take it, was Mitsuda in type, must be nonspecific. The diagnostically valuable reaction occurs in about three weeks. On the other hand, a 48-hour reaction to the Fernandez-Dharmendra lepromin is significant, and of the same diagnostic significance as the three-week reaction obtained with the Mitsuda "antigen." The difference between the two materials is as follows: Mitsuda's lepromin is a crude soup prepared from boiled lepromatous tissue, whereas the Fernandez-Dharmendra lepromin is processed as a bacillary emulsion that has been freed of tissue cells and other debris (Fernandez-J. M. M.: The Early Reaction Induced by Lepromin, Internat. J. Leprosy 8:1, 1940; and Dharemendra: The Lepromin Test, Belra Med. Series 1:1, 1948).

DR. IRWIN I. LUBOWE: A new agent for the treatment of tuberculoid leprosy by topical application has been announced. The Imperial Chemical Industrial Company has incorporated an ethyl mercapatan derivative in an ointment base, which is said to penetrate, and to affect leprosy favorably. The clinical evaluation of this new compound was made at the Nigerian Leprosarium. The agent is trade-named Etisul.

DR. ALEXANDER A. FISHER: The histamine test in this case is reported as "negative." This is confusing unless it is understood that "negative" is to be construed to mean that the normal wheal and flare were obtained, which is not what should occur in a maculoanesthetic lesion of leprosy. On this reasoning, the absence of the flare around the histamine wheal would have been "positive." The words "positive" and "negative," then, are used here in a diagnostic sense, and not in senses of "reaction" and "no reaction."

The best place to do the histamine test in tuberculoid leprosy is at the border of an anesthetic macule. The reaction, if "positive," is then dramatically clear, because the wheal involves both normal and abnormal skin, whereas the flare encircles only that part of the wheal that is on the uninvolved skin, and is absent on that part of the wheal that is on the involved skin.

DR. RICHARD K. SCHER: One of the interesting features of this case is the normal histamine reaction, i.e., both wheal and flare, in three different areas that showed definite cutaneous involvement and nerve damage. The latter was proved by hypesthesia or anesthesia to both pain and heat stimulation. If our diagnosis is correct, then the only explanation is that nerve damage has not proceeded to the point of destruction of the local axone apparatus.

Diagnosis: Skin Changes from Intensive Androgen Therapy Following Bilateral Mastectomy. Presented by DR. CONRAD STRITZLER.

Diagnosis: Multiple Superficial Basal-Cell Epitheliomata with a Change to Deep Invasive Type in One Area. Presented by DR. IRVING D. EHRENFELD.

Diagnosis: Systemic Lupus Erythematosus: Lichen Myxedematosus(?) Presented by DR. HARRY SHATIN.

Diagnosis: Incontinentia Pigmenti? Presented by DR. IRWIN I. LUBOWE.

Diagnosis: Cutis Rhomboidalis with Basal-Cell Epitheliomas: Actinic Skin. Presented by DR. ABRAHAM J. ORFUSS and DR. ALVIN M. LASHINSKY (by invitation).

Case for Diagnosis: Lymphocytic Infiltration? Presented by DR. CONRAD STRITZLER.

CHICAGO DERMATOLOGICAL SOCIETY

Samuel M. Bluefarb, M.D., President
Frederick J. Szymanski, M.D., Secretary

Nov. 19, 1958

Chondritis of the Ears. Presented by DR. STEFAN BIELINSKI.

The patient developed diffuse redness and tenderness of the left ear in April, 1958, followed by marked swelling. The condition persisted, and a month later the right ear was similarly involved. He also had a mild conjunctivitis of the right eye concurrently. Patient was treated by his family physician and an ear specialist because of occlusion of the external auditory canal.

Past history revealed no injury or frostbite.

Physical examination reveals marked thickening of both ears with a dull red erythema. The upper third of the pinna droops, and crepitation can be felt in the cartilage at the line of deformity.

The patient has shown periods of remission and exacerbation of the erythema of both ears in the past two months. He was treated with benzathine penicillin G (Bicillin) injections and warm boric acid compresses and obtained slight improvement. He was later tried on tetracycline (Tetracyn) therapy because penicillin caused pain at the site of injection. In the past month he was given sulfisoxazole (Gantrisin), 1 gm. t.i.d.

Biopsy specimen is presented.

Discussion

DR. THEODORE CORNBLEET: I should like to suggest the possibility of solid edema of the ears.

DR. ALLAN L. LORINCZ: This total type of chondritis of the ears in all probability is a specific entity. There has been a fairly recent report of a rather similar though more acute case by Kahn and Kilbury (*Ann. Otol. Rhin. & Laryng.* 65:615-619, 1956). The peculiar softening of the ear cartilages also calls to mind the interesting phenomenon of wilting of rabbit ears, which can be induced experimentally by intravenous injections of crude papain (*J. Exper. Med.* 104:245-252, 1956).

DR. STEFAN BIELINSKI: In going over the literature I found one case reported in the *Annals of Otology, Rhinology and Laryngology* under the title of "Generalized Chondritis of Both Ears." According to the authors, Kilbury and Kahn, that was the first case of bilateral generalized chondritis of the ears reported. From the description it might very well fit this entity. In that report, biopsy specimens were sent to Drs. Hamilton Montgomery, Pinkus, Helwig, and Rothman. There were various opinions as to the histologic diagnosis. Some thought it could fit into a type of inflammatory reaction, but clinically that did not fit. As in my case, the reported case showed no inflammation. Various therapies were tried, consisting of antibiotics, histamine iontophoresis, and cortisone, with no improvement. My case showed deposits of mucin deep in the corium. I intend to give chymar parenterally to see if it has any beneficial effect.

Subcutaneous Fat Necrosis and Polyarthritis in a Patient with Pancreatitis. Presented by DR. DAVID COHEN and (by invitation) DR. ROBERT SCHOOP and DR. MYRON BERMAN.

The laboratory data on the patient presented are given in the Table.

Discussion

DR. FREDERICK J. SZYMANSKI: Even though this is a rather rare condition it has been my good fortune to see three such cases. The first was a man about 60 with numerous nodules all over the body suggestive of Weber-Christian disease. A biopsy specimen showed massive necrosis of the subcutaneous fat tissue. Soon after developing the nodules, the man died of carcinoma of the pancreas. A few years later, I saw a case similar to the one today, of pancreatitis associated with subcutaneous nodules which on biopsy showed a patchy fat necrosis. It seems incredible that enzymes from the pancreas could enter the blood vessels and then be transmitted to the subcutaneous tissue of the skin. However, the isolation of amylase in the areas of necrosis in this case seems to bear out this premise.

DR. SAMUEL M. BLUEFARB: My colleagues and I reported a similar case from Cook County Hospital. Our patient, a 57-year-old garage mechanic, developed a polyarthritis followed by the appearance of disseminated nodules. The involvement of the pancreas was revealed at autopsy. It was postulated that excessive amounts of circulating enzymes (trypsin and lipase) caused a striking involvement of the extrapancreatic fat tissue, cutaneous and subcutaneous nodules of fat necrosis, and necrosis of the periarticular tissue initiating a chemical polyarthritis.

DR. KURT WIENER, Milwaukee: The phenomenon of fat necrosis brings to mind the fat necrosis occasionally seen after injections of insulin, which, after all, is a pancreatic product.

Submitted for publication March 16, 1959.

Laboratory Data

	10/8	10/9	10/15	10/21	10/23	10/27	10/30	11/4	11/7
Serum amylase, units	165	205	1,272	1,137	620	867	1,468	254	194
Hemoglobin, gm. %	14.5	12.5	12.5	11				12.5	
Hematocrit, %	47	42	40	34				40	
WBC	10,000	8,600	10,000	21,500				7,750	
P, %	82	82	75	81				76	
L, %	9	15	15	11				18	
M, %	1	1	5	1				3	
Bands, %	8	2	5	7				3	
Urine amylase, units		2,664		316					
Aspiration amylase, units					340				
Serum									
Calcium, mg. %		10		9.5				10.7	
Phosphorus, mg. %								4.7	
Albumin, gm. %		4.1	3.9						
Globulin, gm. %		2.2	2.9						
Blood cultures		Negative							
Sulfobromophthalein, %				28.5					
Total bilirubin, %		13.3	7.4					1.2	
Direct bilirubin, %		7.9	4.6					0.5	
LD		291	276						
GOT		100	42						
GPT		75	44						

LD indicates lactic dehydrogenase; normal, 270 units. GOT indicates glutamic oxyacetic transaminase; normal, 40 units. GPT indicates glutamic pyruvic transaminase; normal, 40 units.

DR. DAVID COHEN: This is the second case that I have had the opportunity of seeing. This man came in with a gastrointestinal complaint. In the differential diagnosis liver disease and disease of the pancreas were considered. After a few days he developed an acute erythematous nodular eruption which was thought to be due to the antibiotics but which was due to the systemic condition. A diagnosis of erythema nodosum was made, but the possibility of pancreatitis was kept in mind. We repeated the serum amylase and to our surprise it was 1,200 units. Biopsy revealed a peculiar fatty infiltrate which seems to be diagnostic of this condition. The serum amylase dropped, only to recur whenever any new lesions appeared.

Trypsin seems to have a direct effect on the blood vessels. This may result in the formation of the skin nodules and later the circulating lipase resulted in subcutaneous fat necrosis.

A depression of the blood calcium may develop in extensive pancreatic fat necrosis due to saponification of the fatty acids liberated from neutral fat by the enzymatic action of pancreatic lipase.

Aspiration of one nodule on the leg revealed the presence of serum amylase.

Chronic Lymphatic Leukemia with Secondary Amyloidosis. Presented by DR. FREDERICK J. SZYMANSKI and (by invitation) DR. MARTIN L. GECHT and DR. JACK M. FOX.

Discussion

DR. FREDERICK J. SZYMANSKI: All of us are aware of the association of amyloidosis with multiple myeloma, but the occurrence of amyloidosis in leukemia and lymphosarcoma is rare. A Mexican observer reported some 40 or 50 cases of malignant lymphomas in which he found evidence of amyloidosis in the lymph nodes, kidney, and other visceral organs.

Lichen Fibromucinoidosis (Lichen Myxedematosus). Presented by DR. ALLAN L. LORINCZ and (by invitation) DR. YELVA LYNFIELD.

A 63-year-old shoe repairman of Greek ancestry was in excellent health until early in 1957, when, about a week after a mild "cold," he first noted stiffness in movement of his right thumb. He soon noted that the skin in that area became hardened and thickened in a densely papular manner. These cutaneous changes accompanied by some violaceous redness extended within a few months up the forearm and also commenced in the left hand to extend up that arm. In the ensuing year, the process gradually spread further to involve the skin of the entire upper half of the body and also the upper parts of the legs. The resulting stiffening of the skin has hampered motion of the affected body parts, especially the mouth, face, and upper extremities. A little itching has very occasionally been present, especially on the chest, and some of the affected areas feel numb and slightly tender on pressure.

Difficulty in swallowing began in occasional fashion about 10 months ago and has become persistent during the past 5 months. There has been some loss of appetite but no weight loss or general feeling of illness except for the specific difficulties mentioned. According to the patient's history, corticosteroid therapy was tried about a year ago for approximately three months without success.

On first examination in the dermatology clinic at the University of Chicago, in May, 1958, the skin of almost the entire body, excepting the abdomen and lowermost parts of the legs, showed moderate slightly violaceous erythema and was much thickened, hard, and inelastic. The affected areas were also densely studded with firm, slightly translucent, rounded papules one to several millimeters in size. In some places these papules showed a tendency toward linear arrangement. Hair growth was not disturbed even in severely involved areas, such as the scalp. Sharply outlined, round islands of apparently entirely normal skin up to 3 cm. in size were irregularly scattered through the affected areas on the trunk and extremities. The ears were especially severely affected, with densely clustered very large papules and small nodules. The soles showed diffuse dry hyperkeratosis. The mouth could only be opened to 3 cm., and the tongue which was also stiffened could not be protruded. Mild pitting ankle edema and excessive lacrimation were also present. Physical examination otherwise was not remarkable.

Roentgen-ray examinations of the chest, skull, esophagus, stomach, and duodenum were normal. The hypopharyngeal swallowing mechanism appeared abnormal on fluoroscopy, but this may have resulted from an inability to perform a proper Valsalva maneuver, rather than from infiltration of the hypopharynx.

An electrocardiogram showed only minor changes, suggesting borderline myocardial abnormality. Routine blood cell counts and urinalysis were normal. The Kahn test was nonreactive. Chemical determinations in the blood of urea, glucose, sodium potassium, calcium, phosphorus, chloride, alkaline and acid phosphatases, cholesterol, bilirubin, and uric acid were all normal. Plasma proteins totalled 6.5 gm., with 3.4 gm. of albumin and 3.1 gm. of globulin. The electrophoretic pattern of these proteins was not unusual.

Oral radioiodine study showed 20% uptake after 24 hours, which is normal.

Intradermal test injections of hyaluronidase, trypsin, varidase, heparin, saline, and air failed to alter the lesions. Freezing of a small area with liquid nitrogen resulted in local clearing of the lesions. Intradermal injection of Congo red was followed by unusually rapid clearing away of the dye.

Therapy with large doses of protamine given intravenously and sublingually was unsuccessful, as was oral administration of toluidine blue. At the present time the effects of sublingual administration of varidase on the disease is under study.

Histologic examination of a biopsy specimen from a recent lesion on the right thigh showed areas of decreased density in the upper corium associated with increase in number of fibrocytes. Fine fibers taking metachromatic stain with toluidine blue were present in the same areas. An older papular lesion on the forearm and a specimen from the face showed fibrosis and increased numbers of fibrocytes but much less metachromatically staining material.

There was no increased urinary excretion of polysaccharides. Analysis of affected skin for acid mucopolysaccharide content showed a great increase in this material.

Discussion

DR. ALLAN LORINCZ: Although I have never seen a case of lichen ruber moniliformis, I understand from those who have that it presents a very characteristic picture which differs sharply from this entity of lichen fibromucinoidosis. Even in advanced stages of this latter disease pathologic changes appear largely restricted to the skin. Essentially there seems to be a heavy accumulation of mucin-rich new connective tissue in the dermis. The hardening of the skin is clinically rather suggestive of that seen in scleroderma. However, there occur neither Raynaud's phenomenon nor the esophageal or other internal changes commonly seen in scleroderma. The mucinous material deposited in the skin differs from that which accumulates in various thyroid disorders, as indicated by greater resistance to solubilization by hyaluronidase. These patients have no demonstrable thyroid disease. So far, no therapy has had the slightest success in this unusual disorder.

Syringoma (Eruptive Hidradenomata). Presented (by invitation) by DR. BRIAN POTTER.

A Negro housewife, aged 54, has glaucoma due to granulomatous iridocyclitis (sarcoid?). When seen in 1954, she had hepatosplenomegaly and skin lesions in ear lobes, on chest, arms, thighs, and legs, but parenchymal lung infiltrates seen on x-ray in 1952 had already disappeared. Biopsy specimens of skin lesions on arm and thigh showed a sarcoid granuloma. When seen again, in 1958, the original skin lesions, except for those in the ear lobes, had subsided. There were areas of hyperpigmentation in the previously involved sites and on the face, and general asteatosis. The liver and spleen were no longer palpable.

Two months ago an eruption appeared for the first time on the face. On examination, the forehead and upper part of face present a symmetrically distributed eruption of discrete firm papular lesions.

A biopsy specimen of one of the forehead papules shows numbers of dilated duct-like structures lined by two or three layers of flat cells; some have a comet-like tail of cells.

Syringocystadenoma. Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY and (by invitation) DR. I. GIGLI.

A 29-year-old woman complains of a skin eruption which has been present for 10 years. It started on the forehead as "small bumps" which kept spreading until they covered the entire forehead and cheeks. Condition has been slowly progressive and the lesions are asymptomatic.

The patient presents lesions varying in size from 1 to 3 mm., soft, slightly elevated papules which cover the entire forehead, cheeks, periorbital, and retroauricular areas.

Discussion

DR. DAVID W. KERSTING, Milwaukee: I believe that these are very acceptable examples of syringoma. Noticing Dr. Potter's use of the synonym "eruptive hidradenoma," I recall that Darier used the name hidradenomes eruptifs; and that these multiple lesions sometimes appear rather late in life; for example, recently Woringer and Alt reported multiple syringoma of only six months' duration in a lady 70 years of age (Hidroadenomes Eruptifs, *Bull. Soc. franç. dermat. et syph.* 63:63, 1956).

DR. STEPHEN ROTHMAN: The occurrence of eruptive hidradenomata in a patient with numerous lesions of proved sarcoidosis was a peculiar coincidence. Originally, it was thought that the firm tiny papules could represent micropapular sarcoid.

DR. THEODORE CORNBLEET: In both of these patients the eruption is only on the face, which is somewhat unusual. There is a difference of opinion as to whether the lesions produce sweat. We could not show that they did.

DR. BRIAN POTTER (by invitation): I should point out that our patient does have some lesions of dermatosis papulosea nigra on her face, as well as the syringomas. We could not find anything suggestive of sarcoidosis of the skin now, except possibly the lesion in the left ear lobe.

Tinea and Nodular Granulomatous Perifolliculitis of Legs Caused by T. Rubrum. Presented by DR. ALLAN L. LORINCZ and (by invitation) DR. E. MANFRED BLONER.

A dark-haired, 50-year-old white woman first noted dry scaling of her soles about 15 years ago. Three years ago an initially pruritic eruption appeared on both ankles, slowly extending within a few months over both legs. A similar eruption also developed on the right shoulder and parts of the face and showed periods of extension and regression. Attempts at therapy with a variety of pills and injections of unknown type were said to be unsuccessful.

On examination a few weeks ago in the Dermatology Clinic at the University of Chicago, extensive patches of erythema with active, polycyclic scaling borders were seen on both lower legs posteriorly and extending laterally and anteriorly. Within these areas a number of follicular pustules and inflammatory papules were present. The left great toenail and both fifth toenails were discolored and thickened and showed a subungual debris. On the front and sides of the neck, extending to the face and chest, were less well-defined areas of erythema, with slightly scaling edges and central clearing. Both soles showed dry, fine scaling without erythema.

KOH preparations of scales from leg and chest lesions and of a hair from a follicular pustule all readily showed fungal filaments. Trichophyton rubrum, furthermore, was cultured from scales of the leg lesions, from the left big toenail, and from pus obtained from a leg pustule.

General and family medical histories were noncontributory. The referring local physician had found routine blood cell counts, urinalysis, and fasting blood sugar to be normal.

Additional laboratory tests revealed the serum cholesterol to be 335 mg. % and plasma albumin and globulin and cephalin and thymol flocculation tests to be normal. A 1:200 trichophyton skin test was performed on the left forearm yesterday. There was no immediate urticarial reaction.

Histologic examination of a biopsy specimen taken of a pustular lesion of the right leg revealed follicular purulent inflammation of perifollicular chronic inflammation. Preliminary periodic acid-Schiff stains fail to reveal organisms. Active borders on legs have disappeared or become less conspicuous after local antifungal treatment.

Discussion

DR. IRENE NEUHAUSER: Because of our failure to recognize it, this type of rubrum infection is probably commoner than is believed. In 1954, Wilson, Plunkett, and Gregerson reported 14 cases, and about the same time 14 similar cases were reported in Europe. This condition was first described in this country in 1930 by Harris and Lewis and was probably the same condition described in Europe as Majocchi's trichophytic granuloma.

Since there is an increasing tendency to consider the host rather than the organism, I suggest that this patient be thoroughly studied. I noticed

that the γ-globulin was reported as normal. I have had several γ-globulin estimates made on patients with recalcitrant rubrum infections and the results have been "high level of normal." In spite of this result I treated one patient with γ-globulin and the results were spectacular. Although I do not think γ-globulin is the treatment for these patients, I do think it indicates that there is some component in the serum which is lacking.

DR. ALLAN L. LORINCZ: It has been suggested by early observers of this condition that predominant involvement of the legs of dark-haired women may be a consequence of traumatization incidental to frequent and vigorous shaving. As pointed out by Dr. Neuhauser, it is clear that unknown factors of individual susceptibility must lie behind these severe T. rubrum infections. We have observed unusually widespread or severe T. rubrum infections, particularly in Cushing's syndrome, lymphomas, severe liver disease, and hypothyroidism. Of course, they may also occur, as in this case, in apparently normal persons. On very rare occasions, T. rubrum infections may completely overwhelm whatever natural defense mechanisms normally operate and produce fatal systemic disease involving almost all internal organs.

As concerns therapy of the type of case presented here, Wilson and his associates obtained good results with a combination of local treatment with Asterol ointment and x-ray irradiations and systemic administration of iodides (*Arch. Dermat. & Syph.* 69:258, 1954).

Although in vitro psoralen-ultraviolet treatment powerfully inhibits the growth of T. rubrum, our experimental trial of psoralen administration combined with local ultraviolet irradiations was therapeutically unsuccessful as in this case.

Alopecia Cicatrisata. Presented by DR. ARTHUR L. SHAPIRO.

Case for Diagnosis: Scars. Presented (by invitation) by DR. ROGER W. PEARSON, DR. ARNOLD COMBINER, and DR. M. BARRY KIRSCHENBAUM.

Case for Diagnosis (Localized Periarteritis Nodosa?). Presented by DR. LOUISE E. TAVS and (by invitation) DR. PAUL V. SCHMITT.

Alopecia Areata? Seborrheic Dermatitis? Presented by DR. ALFRED FALK and (by invitation) DR. WILFRED SCHMEROLD and DR. ROLAND S. MEDANSKY.

Alopecia Mucinosa. Presented by DR. ALFRED B. FALK and DR. FREDERICK J. SZYMANSKI.

A girl, aged 10 years, was well until August, 1958, when she developed a swelling on the left side of the chin. On examination, the swelling was found to be a plaque which could be palpated easier than seen, measuring about 1.5 cm. in diameter and about 0.5 cm. thick. There were no subjective complaints.

A specimen of skin was removed with the following findings which are characteristic of the condition.

Histopathologic report: Microscopic examination reveals a peculiar foamy-like degeneration of the hair follicles. Follicular cells are separated by a very faint homogenous substance that appears edematous. There is a moderately severe inflammatory infiltrate involving some of the hair follicles, sweat glands, and superficial vessels. Special stains (Best's mucicarmine, Gomori's aldehyde fuchsin, and Rinehart-Abul-Haj) revealed a mucinous-appearing substance in two to three follicles in each section. Diagnosis: Alopecia mucinosa.

Discussion

DR. DAVID W. KERSTING, Milwaukee: I rise only to thank, and, if I may, compliment the presenters for making this diagnosis and bringing an example of this condition so dramatically to our attention. Dr. Pinkus described alopecia mucinosa just last year (A.M.A. ARCH. DERMAT. 76:419-426, 1957). Dr. Szymanski's histological work-up today is very enlightening, showing so well this pooling of epithelial mucin in the follicle, a "mucinosis of the follicular sheath," as the late Dr. N. P. Anderson termed it (A.M.A. ARCH. DERMAT. 76:425, 1957). I am anxious to learn more about the special stains which Dr. Szymanski has employed so well in this case.

DR. FREDERICK J. SZYMANSKI: Alopecia mucinosa is a condition described recently by Pinkus. According to his description, alopecia mucinosa occurs on the scalp, eyebrows, face, chin, and forearms. Alopecia develops when the lesions are located on the hairy areas, such as the scalp and eyebrows. Clinically, the condition may appear as indurated nodules, either single or multiple. Cases have simulated lichen spinulosus. Both children and adults are affected by this condition. The microscopic alterations are striking and seen once are not easily forgotten. The process begins in the sebaceous glands as an edema which separates the cells. The hair follicle itself becomes enlarged with an edematous foamy appearance. A mucinous substance separates the cells of the hair follicle.

As far as treatment is concerned, alopecia mucinosa often clears spontaneously or it responds to a very small amount of x-ray. Dr. Pinkus claims this condition may be an infectious disease, probably viral in nature.

Lichen Nitidus. Presented by DR. SAMUEL M. BLUEFARB and DR. SILAS WALLK and (by invitation) DR. RICHARD I. BERNSTEIN.

A 16-year-old white girl noted papular nonpruritic eruptions appearing on her wrists and palms five years ago. When seen by her family physician soon afterward, similar lesions were discovered in the axillae, on the knees, and on the upper inner aspects of her thighs. At that time,

the patient began to have thickening and scaling of the palms. There is no one in the family who has any dermatologic history of similar nature.

Her lesions consist of multiple nonconfluent, discrete, umbilicated papules. There is noted a slight thickening of the palmar skin. A histologic section of the lesion is presented.

Discussion

DR. H. M. SHAIR, Quincy, Ill.: The case at Cook County Hospital showed stippling of the palms, a change which I first saw in a patient with lichen nitidus one year ago. The nondescript papules on the distal phalanges are lichen nitidus, too, but are undiagnosable, except for the characteristic eruption elsewhere.

In regard to the prognosis in lichen nitidus, one patient, seen nine years ago, is now entirely free of the eruption (Case 2 reported by Pinkus and Shair, *A.M.A. Arch. Dermat. & Syph.* 65:82-87 [Jan.] 1952); a second patient, seen one year ago, is now "50% better."

DR. S. J. ZAKON: I should like to call to the attention of our younger colleagues that it was Felix Pinkus (1868-1950) who described lichen nitidus in 1907 and that our contemporary Herman Pinkus described alopecia mucinosa.

DR. STEPHEN ROTHMAN: Herman Pinkus was not born when Felix Pinkus described lichen nitidus.

DR. SAMUEL M. BLUEFARB: The interesting feature in our case are the palmar lesions. Lichen nitidus may develop lesions on the palms, but, in our experience, this is not a common site.

Sickle-Cell Ulcers (in Sickle-Cell Trait Patients). Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY and (by invitation) DR. I. GIGLI.

A 13-year-old Negro girl noticed the appearance of a small ulceration on the anterior medial aspect of the left leg three months ago. She is 5 ft. 8 in. tall, thin, with long fingers and a long narrow face. She presents bilateral ulcerations of the legs.

Hemoglobin was 12.7 gm. (74%). Serum electrophoresis showed sickle-cell trait pattern. Sickle-cell preparation was negative.

An 11-year-old girl was seen for the first time in our clinic with a history of chronic leg infection (ulcers) of two months' duration. She received local treatment with very little improvement. However, the ulcers healed in late October.

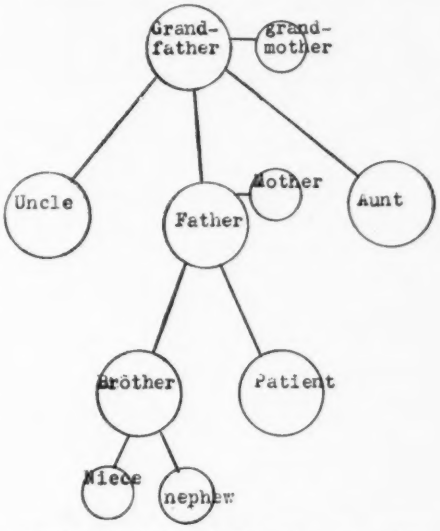
Hemoglobin was 10.6 gm. (60%). Serum electrophoresis showed sickle-cell trait pattern. Sickle-cell preparation was positive.

Discussion

DR. BERNARD YAFFE: Serum electrophoresis has revealed new findings in sickle-cell anemia. Not only can patients with anemia and sickling phenomenon in the peripheral blood develop clinical signs, but patients with the inherited trait can have leg ulcers, anemia, and hemorrhages in the spleen. This trait is revealed by the presence of abnormal hemoglobins in the electrophoretic pattern. Apparently, another pathologic mechanism is at play other than tamponade of arterioles.

DR. SIDNEY BARSKY: These cases represent a little more than sickle-cell trait. The habitus of these patients and the lower hemoglobin in the first case indicate that these might be borderline cases rather than sickle-cell trait. Eventually, they may go on to develop sickle-cell anemia.

Peutz-Jeghers Syndrome. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. J. LATONI.



Genetical illustrations including four generations. Large circles indicate affected members; small circles, unaffected members.

Discussion

DR. KURT WIENER: It is amazing that as late as 1921 the first eight familial cases were described (Peutz) and that it took another 20 years until Jeghers renewed the interest in this striking combination, which follows a dominant pattern of heredity. Since then cases have been presented frequently. Kitamura et al., of Tokyo, recently emphasized the pigmentations of palms and soles which one of our patients has. There is a nonfamilial type of diffuse polyposis of the colon, which appears late in life. These cases have not the ink-spatter type of perioral pigmentation but rather chloasmatic and diffuse brownish discolora-

tion in the face. At least two families with polyposis coli and familial multiple sebaceous cysts have become known.

DR. SAMUEL W. BECKER JR.: The pigmented lesions on the lips and hands in the Peutz-Jegher syndrome are lentigines. Lentigines may occur without associated polyposis. If a patient has lentigines and complains of abdominal distress then a search for polyps of the small bowel should be made. If a patient has lentigines without abdominal symptoms then the laboratory tests are not indicated.

DR. STEPHEN ROTHMAN: We have shown here patients with Peutz-Jeghers syndrome, and I believe this condition is not uncommon. It is true, however, that different kinds of nevoid macular pigmentation is much more common in Japan. Dr. Kitamura, in Tokyo, has described and classified them beautifully. While lentigines occur very commonly on the skin, their occurrence on the vermilion border is rather rare. In such cases one should rule out the simultaneous presence of polyposis.

DR. SAMUEL M. BLUEFARB: Pigmentation of the lips was first reported by Hutchinson, in 1896. Peutz, in 1921, first described a syndrome of heredity, pigmentation, and polyposis, and later Jeghers, in 1949, again called attention to the intestinal polyposis. Our patient had, in addition, other congenital anomalies, such as a cervical rib and a congenital heart disease. A dominant pleiotrophic gene with penetrance seems to be the cause of this disorder.

Metastatic Lesion to the Umbilicus. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. RICHARD I. BERNSTEIN.

Discussion

DR. SAMUEL M. BLUEFARB: Schiebel (J.A.M.A. 157:1489, 1955) reported five cases of metastasis to the umbilicus from an underlying malignancy. In three of his patients the primary lesion was in the stomach, and the ovary was the site of the primary lesion in the other two patients. It is our impression that our patient has carcinoma of the ovary.

Pityriasis Rubra Pilaris. Presented by DR. JULIUS E. GINSBERG and (by invitation) DR. CHRISTINE SUTTON.

Lichen Nitidus. Presented (by invitation) by DR. ROGER W. PEARSON and DR. YELVA LYNFIELD.

Incontinentia Pigmenti. Presented by DR. DAVID V. OMENS and (by invitation) DR. HAROLD D. OMENS and DR. CHESTER L. KROLL.

Incontinentia Pigmenti. Presented by DR. DAVID V. OMENS and (by invitation) DR. HAROLD D. OMENS and DR. J. LATONI.

Dermatofibrosareoma Protuberans. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. J. LATONI.

Peutz-Jeghers Syndrome. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. RICHARD I. BERNSTEIN.

Unilateral Hyperkeratosis Palmaris (Verruca Vulgaris, Keratoderma?) Following an Accident. Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY, and (by invitation) DR. I. GIGLI.

Unilateral Excoriations Associated with Cerebrovascular Accident. Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY, and (by invitation) DR. RICHARD I. BERNSTEIN.

Von Recklinghausen's Disease with Clubbing of the Fingers of Undetermined Etiology. Presented by DR. DAVID V. OMENS and (by invitation) DR. HAROLD D. OMENS and DR. RICHARD I. BERNSTEIN.

Sarcoidosis. Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY, and (by invitation) DR. J. LATONI.

Cold Urticaria. Presented by DR. THEODORE CORNBLEET, DR. SIDNEY BARSKY, and (by invitation) DR. J. LATONI.

Melanotic Melanoma. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. CHESTER L. KROLL.

Basal-Cell Epithelioma, Radiodermatitis, and Squamous-Cell Carcinoma of Lip. Presented by DR. SAMUEL M. BLUEFARB, DR. SILAS WALLK, and (by invitation) DR. CHESTER L. KROLL.

Rodent Ulcer. Presented by DR. DAVID V. OMENS and (by invitation) DR. HAROLD D. OMENS and DR. CHESTER L. KROLL.

Case for Diagnosis (Gumma? Phagedenic Ulcer?). Presented by DR. THEORDORE CORNBLEET, DR. SIDNEY BARSKY, and (by invitation) DR. I. GIGLI.

Annual Meeting, Jan. 21, 1959

Sarcoidosis?—Leukodermia? Presented by DR. SAMUEL M. BLUEFARB and DR. SILAS WALLK, and (by invitation) DR. J. LATONI.

A 24-year-old Negro woman developed tender depigmented, macular, infiltrating plaques of various shapes and sizes on the lower extremities seven months ago, followed by similar lesions on the upper extremities and trunk. There is no involvement of the lacrimal glands, the conjunctiva, the lymph nodes, or the mucous membranes of the nose. No spina-ventosa-like enlargement of the fingers or toes is observed. No hepatosplenomegaly is present.

Laboratory Findings.—Complete blood count and differential normal. Blood chemistry: protein, total, 8.1 mg. %, albumin 4.4 mg. %, globulin 3.7 mg. %, calcium 10.7 mg. %, alkaline phosphatase 3.0 Bodansky units, gamma globulin 1.6 mg. %. Tuberculin test, 1:1,000, negative. X-ray plates

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of chest, skull, and extremities were reported normal. A biopsy is present.

Sarcoidosis with Granulomatous Uveitis. Presented by DR. SAMUEL M. BLUEFARB and DR. SILAS WALLK, and (by invitation) DR. J. LATONI.

The patient, a 55-year-old Negro man, first developed skin lesions four years ago on his lower extremities, followed three and one-half years later by involvement of the upper extremities, chest, back, and face. The patient has been complaining of dyspnea and of some visual impairment for the last six months.

Physical examination reveals a hypertensive patient (222/130) who presents generalized monomorphous, asymmetrical dermatosis, consisting of round, firm, reddish-purple, papular and nodular lesions, movable over the underlying tissues, and varying in size from 1 to 6 mm. Moderate lymphadenopathy is noted, more prominently in the epitrochlear and supraclavicular areas. Examination of the abdomen reveals hepatosplenomegaly. No osteitis or granulomatous changes are observed on the fingers or toes, and the mucous membranes of the nose are apparently normal.

An eye examination, performed on January 16, 1959, reveals normal tension of both eyes, large right pupil, right corneal endothelial surface, and keratic precipitates on the right iris; lens and vitreous, intact and clear.

Laboratory Findings.—Blood: hemoglobin 87%, white cells 2,950, red blood cells 3,600,000.

Blood Chemistry.—Fasting blood sugar 104 mg. %, B.U.M. 12 mg. %, total protein 8.1 mg. %, albumin 4.3 mg. %, globulin 3.8 mg. %, chloride 102 mg. %, sodium 138 mg. %, potassium 4.3 mg. %, alkaline phosphatase 6.8 mg. %, acid phosphatase 0.1 mg. %, cephalin flocculation 21 mg. %, thymol 4.1 mg. %, gamma globulin 1.57 mg. %, B.S.P. 140 mg. %, cholesterol 400 mg. %, tuberculin, 1:10,000, negative. X-rays of chest and extremities normal. Electrocardiogram showed left heart strain, not diagnostic. A slide is presented.

Sarcoidosis. Presented by MAJOR HENRY L. HINES, Chanute Air Force Base, Ill., and DR. MALCOLM SPENCER, Danville, Ill.

This 21-year-old Negro airman was first seen in the Dermatology Clinic October 29, 1958, with a history of noting small hard nodules under the skin of the arms and legs since October 20, 1958 (nine days' duration).

Past history was negative, except for chronic recurrent urethral discharge, diagnosed as gonorrhea on three occasions, and treated with penicillin. Last course was in May, 1958. Two weeks previously, the patient had been under treatment for pleuritic chest pains and symptoms of upper respiratory infection.

On physical examination, there were multiple pea-sized subcutaneous nodules, mainly on the arms and legs, and generalized lymphadenopathy—cervical, axillary, and inguinal. Epididymis was enlarged and tender on right side.

Laboratory Findings.—Chest x-ray showed hilar adenopathy, no parenchymal lesions. Skeletal survey of small bone x-rays negative. Biopsy of skin nodule: sarcoidosis (AFIP); biopsy of cervical node: sarcoidosis (AFIP); biopsy of epididymis: sarcoidosis (AFIP).

Blood: white cells 4,500, hemoglobin 16.6, hematocrit 54%, sedimentation rate 19 mm/hr. Total protein 8.7 mg. %, albumin 4.6 mg. %, globulin 4.1 mg. %, ratio 1:1/1. Serology negative. Urinalysis normal. Sperm count 0; P.S.P., 50% excretion in 2 hours. P.P.D. No. 1, negative, P.P.D. No. 2, negative. Alkaline phosphatase 7.8%.

Eye consultation: enlarged infiltrated lacrimal gland, right eye; no uveitis or parotid gland enlargement. Thymol turbidity 3.2 units; cephalin flocculation 3+, 24 hours; 3+, 48 hours. P.S.P. 27.5% retention. Gamma globulin 30.2% (normal 12%-18%).

Diagnosis: systemic sarcoidosis (Boeck) with sarcoidal epididymitis, subcutaneous nodules, and pulmonary hilar adenopathy.

Case for Diagnosis: Panuveitis and Papular Eruption. Presented by DR. ADOLPH ROSTENBERG JR., DR. PAUL GRIFFITH, and DR. SIDNEY BARSKY, and (by invitation) DR. ROLAND S. MEDANSKY of Hines Veterans Hospital.

Discussion

DR. HENRY E. MICHELSON, Minneapolis: Most of the things about the condition have been discussed here many, many times, but what interests me most of all now is the presenting symptom. It is quite simple in the case of dermatologists who see a skin lesion and then analyze it, but we wonder what makes the people in other specialties become suspicious that the patient may have sarcoidosis. The ophthalmologists, I believe, always have sarcoidosis in mind when the patient has inexplicable uveitis, and I believe internists have their attention drawn to the condition mostly through x-ray examination of the chest, and isolated glandular enlargements that are asymptomatic.

One must keep in mind that the most obscure symptom may be due to sarcoidosis. For example, I read a recent report where the only finding was albuminuria, but biopsy of the kidney revealed sarcoidosis.

To come to the specific cases of today, I do not believe I have ever seen leukodermia due to sarcoid before. We have always considered leukodermia as occurring commonly in syphilis, leprosy,

postpsoriatic or postfungous infection, and in pityriasis lichenoides chronica. I believe this is the first patient I have seen with leukodermia in sarcoidosis. The patient with the small lichenoid lesions presents an unusually widely-spread example, and it is the kind that is seen in colored people, but rarely in the white race.

As far as treatment is concerned, I do not believe that patients with sarcoidosis should receive steroids, unless there is definite indication which is not the mere presence of the disease.

DR. S. J. ZAKON: I recently presented, at a staff meeting of Mount Sinai Hospital, a young colored woman with sarcoidosis of the face and the extremities, with a marked and bizarre leukoderma. One of our pathologists, who worked for five years in India, suggested the diagnosis of leprosy, even though the histology was definitely that of sarcoidosis. This only illustrates that in India they have a high index of suspicion for leprosy, and justifiably so.

DR. J. B. HOWELL, Dallas, Texas: I would like to direct attention to the scar on the nose of Dr. Rostenberg's patient with sarcoidosis. The history may be incorrect, but the patient was of the opinion that one of his first lesions was an annular lesion on the nose, which was treated for skin cancer with x-ray therapy. A specimen for biopsy was not taken. An isolated superficial annular lesion of sarcoid can clinically mimic basal-cell cancer. I have seen a few patients with sarcoid that have been treated for basal cell carcinoma, and I wonder if this could have happened to the nose lesion.

MAJOR HENRY L. HINES, Rantoul, Ill.: I want to mention briefly two things which I learned from this case. One is the necessity of doing multiple biopsies. This was clearly brought out in Dr. Harold Israel's report of his experiences with 160 cases, in the Archives, 1958. (Israel, H. R., and Sones, M.: Sarcoidosis: Clinical Observation on 160 Cases, *A.M.A. Arch. Int. Med.* 102:766, 1958.)

I had quite an argument with the Department of Internal Medicine and Surgery when I insisted upon lymph node biopsy in addition to the skin sample taken. The lymph nodes were also positive. Because of the epididymal lesions, which were also biopsied and found to be compatible with sarcoidosis, the urologist became interested, and did a cystoscopic examination to look for bladder infiltrates called malakoplakia. I had previously not been aware of these lesions as part of the sarcoidosis syndrome. Apparently the urologists are more acquainted with this than the dermatologists.

Pemphigus Erythematosus. Presented by DR. IRENE NEUHAUSER and (by invitation) DR. JACK M. FOX.

Discussion

DR. LOUIS BRUNSTING, Rochester, Minn.: It is my conviction that pemphigus erythematodes and the Senear-Usher syndrome are early signs of pemphigus foliaceus, but I also believe that pemphigus foliaceus is distinct from pemphigus vulgaris. Patients with pemphigus foliaceus manifest features of hypersensitivity and react poorly to stress. The skin condition is usually aggravated by the application of greases. The response to treatment with steroids and antimalarials is irregular. On the other hand, the prognosis is better than in cases of pemphigus vulgaris.

DR. DAVID V. OMENS: All the cases we had seen at Rush Medical College and Cook County Hospital eventually died from pemphigus.

DR. IRENE NEUHAUSER: In 1949, Dr. Senear and Dr. Kingery published an article in which they reviewed all published cases to date. They came to the following conclusions: Cases which present the combination of elements suggesting lupus erythematosus, seborrheic dermatitis, and pemphigus, in the majority of instances prove to be pemphigus erythematodes. Those which show a histological picture of lupus erythematosus should be excluded. In some cases, the lupus-erythematosus-like element may predominate; in others, the seborrheic, and in still others, the pemphigoid. There were also elements suggesting psoriasis, impetigo, and eczema, in different cases or at different stages. They concluded that, although it was originally thought that pemphigus erythematodes was a benign type of pemphigus, experience had shown that a fatal outcome was not uncommon once the clinical picture of pemphigus vulgaris or pemphigus foliaceus had developed. They also stated that in some cases the picture of pemphigus vulgaris or pemphigus foliaceus preceded the picture of pemphigus erythematodes. Finally, they said there was no justification for the term "Senear-Usher syndrome"; it should be known as pemphigus erythematodes.

A few years ago, after returning from Spain, where there seemed to be a great deal of controversy, I asked Dr. Senear to tell me very definitely his opinion of the syndrome. He said pemphigus erythematodes was a clinical picture which might occur in either pemphigus vulgaris, pemphigus foliaceus, or fogo selvagem. He further stated that one's impressions were influenced by the areas in which one practices, different types predominating in different areas. Dr. Senear did not think that the Brazilian type of pemphigus foliaceus-fogo selvagem was related to pemphigus vulgaris or pemphigus foliaceus, but was probably of infectious origin.

I recently visited a hospital in Southern Brazil where only patients with fogo selvagem were

treated. Here I found the most typical picture of pemphigus erythematodes which I have ever seen.

Erythema Induratum? Presented by Dr. Sidney Barsky and (by invitation) Dr. I. Gigli.

This 19-year-old Negro girl was well until three weeks prior to admission, when she noticed "dark spots" on the anterior aspect of both legs. These spots were noted to be firm to palpation. There were no constitutional symptoms.

On examination, the patient was found to be well-developed and well-nourished. The skin lesions consisted of several deep indurated, hyperpigmented, nontender areas, measuring approximately 5 mm. in diameter, on the anterior aspect of both legs. There were a few such lesions on the posterior aspect of the legs. There were no other positive physical findings.

Laboratory Findings.—Urinalysis was normal. Blood: hemoglobin 85% (13.2 gm.), white cells 8,800. Serology was negative. Twenty-four hour sputum examination showed no acid-fast bacilli. Blood chemistry: Total protein 7 mg. %, albumin 4.4 mg. %, gamma globulin 1.05 mg. %. Chest x-ray normal. Mantoux test 1:10,000 ++. Biopsy is presented.

Discussion

Dr. Henry E. Michelson, Minneapolis: I have been interested in erythema induratum for a long time, as you all know, and I am convinced that in this country a very, very small percentage of patients with that clinical finding do have the tuberculous form of erythema induratum. In this particular patient, I do not see how we can attribute the condition to be due to tuberculosis, for the findings in that direction are all negative. I think panniculitis of the Makai type should be considered. Panniculitis again is not a diagnosis, and I think our view about that condition will change. One may look upon panniculitis somewhat as we do upon erythema nodosum. It may be an accompanying symptom of a lot of underlying conditions.

Dr. Otto Stegmaier, Davenport, Iowa: I would like to disagree with Dr. Michelson. If you follow these patients with recurrent ulcerations of the legs, they recur in the winter and heal in the spring, and many of them will develop a tuberculous node in the neck. If tested to old tuberculin, many of them will be positive to the 1:1,000,000. If you follow these patients for a long time, other evidences of tuberculosis will appear.

Pseudoxanthoma Elasticum. Presented by Dr. Samuel M. Bluefarb and Dr. Silas Wallk, and (by invitation) Dr. I. Gigli.

The patient, a 58-year-old Negro woman, was admitted to Cook County Hospital, and diagnosed as pseudoxanthoma elasticum. On admission, she was complaining of a "skin rash" of six months' duration, localized on the neck, arms, and in both inguinal areas. There was a history of frequent epistaxis, weakness, and loss of weight. The skin was hard and infiltrated with fine, white, calcific deposits. After one course of sodium Versenate the patient feels better and the skin is more loose, even though she still has calcium deposits. She has had no more epistaxis.

Laboratory Findings.—Before treatment, urea clearance was 15%, calcium (gm/100) 11.1, calcium in 24-hour urine 41.8 mg/vol. The patient was given 2 gm. sodium Versenate daily for five days, and the urea clearance was 27.5%, calcium in urine 124.25 mg/vol., calcium in blood two hours after Versenate was 9 mg/100, and 4 hours after Versenate was 9.9 mg/100. After five more days of treatment, with 2 gm. daily, the urea clearance was 41%, calcium in 24-hour urine 190 mg/vol., and calcium in blood 9.8 mg/100 and 11.2 mg/100. After five more days of treatment, urea clearance was 46%, 24-hour calcium in urine 182 mg/vol., and calcium in blood was not done. The patient was getting a control diet of 1.2 gr. (about 1 mg.) of calcium daily.

Discussion

Dr. Francis W. Lynch, St. Paul: This patient was most interesting to see, because of the classical picture of the disease. My particular interest was in the use of chelation in the attempt to withdraw the calcium from the involved areas. We had considered similar therapy in a less extensive example of pseudoxanthoma elasticum, but the internists discouraged us because of their fear that any calcium in the vessels might be serving as a supportive structure, and its removal might lead to weakening of the vessel walls and possible disastrous results. I am glad to note the very satisfactory result in this patient presented today.

Dr. Zachary Felsher: I was interested in what the exact pathology of pseudoxanthoma elasticum is. A few years ago, some English workers found, on electron microscopy, that it did not seem to be the elastic fibers themselves that were involved. It seemed to be some change in the collagen. The collagen fibers were shorter, and apparently these were the fibers that were taking the elastic stain, this being analogous to the condition supposedly seen in senile elastosis. Apparently the collagen fibers undergo degeneration. In pseudoxanthoma elasticum, apparently, the elastic stain is taken up by these fibers, according to these English investigators.

Dr. Marcus R. Caro: I have been interested in the problem of pseudoxanthoma elasticum, and recently Dr. Szymanski and I reported a case like this. Because of the presence of calcium in the

degenerated fibers of pseudoxanthoma elasticum, we had the von Kossa stain performed, and in all cases of pseudoxanthoma elasticum the stain was taken up in the middle portion of the corium. I cannot recall seeing a single case of senile elastosis in which calcium was found demonstrable by this stain. If I remember correctly, it was my impression that the enzyme, elastase, causes absorption of all the degenerated fibers in pseudoxanthoma elasticum, and this feature suggested that the two are entirely dissimilar.

DR. CHARLES C. CACCAMISE, Jackson, Tenn.: I recently saw a 35-year-old woman with pseudoxanthoma elasticum. She was losing the sight in her right eye. She had anginoid streaks. The complication was in the eye, and nothing could be done about it.

DR. SAMUEL M. BLUEFARB: There is an increase in the deposition of calcium phosphate in the degenerated elastic tissue in pseudoxanthoma elasticum. The thought occurred to us to try treating this patient with a chelating agent, such as sodium Versenate. However, our patient had only a 15% renal function, and we were advised "to go easy" with this therapy. This patient was given three courses of sodium Versenate, and we were able to increase her kidney function from 15% to 46%. In addition, the epistaxis and gastrointestinal bleeding have ceased, and the patient feels better. Although we have not helped the basic process in pseudoxanthoma elasticum, we have improved our patient's physical condition by increasing her kidney function, stopping her hemorrhages, and bringing about a general symptomatic improvement.

Lympangioma Circumscripta Cutis. Presented by DR. SIDNEY BARSKY and (by invitation) DR. CHESTER L. KROLL.

This patient is an 11-year-old Negro girl. At 11 months of age, she had a "tumor" removed surgically from the abdominal wall of the right lower quadrant. About nine months ago, small brown-gray "bumps" began to appear around the incisional scar. These nodules have increased both in size and number. The patient has pruritus in this area.

The skin of the abdominal wall in the right lower quadrant shows brown-gray verrucous-like lesions, arranged irregularly in groups around the incisional scar. When one of these lesions was punctured, a clear fluid exuded.

There are two slides presented. One slide represents a section from the original tumor. The other slide is from a recent biopsy specimen.

Discussion

DR. OTTO STEGMAIER, Davenport, Iowa: Recently, I saw a case in which a deep lesion appeared first, and, after a number of years, a superficial lesion developed. It is of interest that lympangioma is just the opposite of hemangioma. In the way of treatment, there is not much that can be done. In the case shown today, the lesion is segmented, and apparently does not cause much trouble; therefore, it should be left alone.

DR. MARCUS R. CARO: I should like to comment on two points. First, the patient had a tumor removed at 11 months of age, which was diagnosed as hemangioma. This may have been a hemangiolymphangioma, and the present tumor may represent a local recurrence.

As to therapy, I do not think that these lesions should be let alone. They are very superficial, and sooner or later the patient is going to bleed. I have treated a number of such lesions with dry ice applied with pressure, and have obtained good results.

DR. LEON GOLDMAN, Cincinnati: I did not see the case, but we are interested in the surgical treatment of lymphangioma circumscriptum. However, even after good surgery there are recurrent lesions. In the superficial type with papular lesions, we can prick some with a needle and make a differential diagnosis. For the deep lesions, one should palpate very carefully to determine the extent. The surgeon who operates for this type should go very wide and deep, because of the frequency of recurrence with inadequate removal.

Nevus of Ota. Presented by DR. DAVID V. OMENS, and (by invitation) DR. HAROLD D. OMENS and DR. J. LATONI.

The patient, a one-year-old female baby, has had the presenting condition since birth. The lesion has gradually lost some of its original pigmentation. No subjective symptoms are present, and no other members of the family are affected.

Physical examination reveals a single light blue to steel blue, irregular, diffuse, macular patch, involving the frontal, ocular, circumocular, and infraorbital areas.

Discussion

DR. SIDNEY J. KAPLAN, Waukegan, Ill. (by invitation): I had a most interesting experience relative to nevus of Ota. A lay jury was asked to decide whether a nevus of Ota had resulted from injury to the eye. The woman's eye had been traumatized by a fist, and developed a permanent pigmentation. Slides stained for iron failed to show the presence of hemosiderin. The diagnosis of nevus of Ota was confirmed by two histopathologists. In preparation for the court appearance, I found a report in the Los Angeles Dermatological Society transactions of four cases of nevus of Ota which appeared after trauma.

The lay jury decided in favor of the plaintiff, on the basis that the nevus of Ota resulted from the trauma.

DR. MARCUS R. CARO: Dr. Kersting and I recently reported a case in which the nevus of Ota had been present unchanged, and followed by us for 24 years.

DR. SAMUEL W. BECKER JR.: It is unfortunate that many lesions depend on the presence of pigment for diagnosis. It is almost certain that the human skin contains melanocytes in the dermis. Normally, these cells contain no melanin. The blue lesions, nevus of Ota, blue nevus, mongolian spot, have been described most frequently in races which produce a lot of melanin. These lesions may be fairly frequent in light-skinned races, but would be missed because the lesions contain little melanin.

Dermatomyositis. Presented by DR. ADOLPH ROSTENBERG JR. and (by invitation) DR. BETSY BECKER BRENNAN.

This 10-year-old white boy was well until September, 1957, when he developed a "severe cold." This was followed by a "burning pain" in the anterior part of the neck, and by the development of redness of the face which has persisted. During the following winter, the boy developed "chapping" of his fingers. In April, 1958, the patient became weak, and noticed generalized muscle soreness. Muscle weakness became so severe that he was unable to walk. He was hospitalized at this time, and a complete examination, including muscle biopsy, revealed no abnormalities. The child was then taken to Bobs Roberts Hospital, where a clinical diagnosis of dermatomyositis was made, and treatment with Aristocort (5 mg. q.i.d.) was started. Since this time, several different types of steroids and ACTH gel have been used in treating this boy, but he has shown only slight improvement. He is now taking cortisone, 10 mg. b.i.d.

Physical Examination.—The face shows a purple-red erythema, and a slight scaling over the forehead, eyelids, cheeks, nose, and chin. There is a "moon face," probably developing as a result of steroid administration. There is similar erythema and scaling on the dorsum of the right hand. The knuckles of both hands show small lesions, which show a slight purple-red erythema. There is marked muscular weakness and tenderness, most marked in the proximal parts of the extremities. There is also involvement of the trunk muscles.

Laboratory Findings.—Urinalysis shows a few red blood cells and white blood cells, and casts. Blood: hemoglobin 11 gm., white cells 4,000-6,000, differential normal except for 10% monocytes; sedimentation rate 28 mm. (uncorrected). L.E. preparation negative. Latex agglutination (for rheumatoid arthritis) negative. Bone marrow normal. Serum: proteins normal, N.P.N. normal, electrophoresis normal, calcium 4.5 mg. % (normal 5-6 mg. %), alkaline phosphatase 1.6 mg. % (normal 5-15 mg. %), inorganic phosphorus 2.6 mg. % (normal 1.7-3.2 mg. %), transaminase 38 mg. % (normal 8-40 mg. %). Twenty-four hour creatine 3.4 mg/kg. (normal 4.2 mg/kg.), creatinine 2.9 mg/kg. (normal 0.7-2.0 mg/kg.). Electrocardiogram within normal limits.

Histopathological Report.—A biopsy was performed on a gastrocnemius muscle and the overlying skin. Skin was essentially normal. Muscle: There are numerous perivascular foci of lymphocytic infiltration. The nuclei of the muscle fibers and of the sarcolemma are quite prominent.

Impression: Mild chronic myositis consistent with dermatomyositis.

Discussion

DR. HEYWORTH SANFORD (by invitation): I have been studying this boy for some weeks. The child has been sick for over a year. I suddenly find that a diagnosis has been made. I do not consider this to be a case of dermatomyositis, because to me it does not conform to the original description made by Jacoby in 1908. There are three cardinal symptoms: characteristic skin lesions, muscle pain, and indurating edema. This boy has never had any muscle pain, or flexion. The rash as originally described was of the butterfly type like lupus, which started on the face, and spread to the rest of the body as a maculopapular eruption. This boy has never shown this type of eruption, and there never has been any edema. His skin does have the doughy feeling, but appears perfectly normal. He does show some muscle weakness. Probably we are all talking about the same thing, and it is quite possible that he has some type of collagen disease that does not conform to my idea of dermatomyositis. I have always felt that dermatomyositis is a dermatological condition, and you dermatologists have allowed the internist to place it under collagen diseases.

Histologically, you will find a peripheral infiltration of round cells around the blood vessels similar to those seen in periarteritis nodosa, as well as degenerative changes in the interstitial connective tissue. Also, there is an edema of the vessels of the corium, which you will also find in lupus and in scrofula. In other words, you have an inflammatory and degenerative change due to colloid degeneration. I imagine it is only a matter of form as to whether you call this dermatomyositis or something else. I have been considering this boy as lupus, but have been unable to find any LE cells. There are cases, however, in which you do not find these cells.

There is one other point that I would like to mention. As you probably know, when these individuals die, their tissues are swarming with bacteria—usually streptococci. One boy I saw in the 1930's, at the time that the sulfanilamides were first introduced, was given prontosil, and was alive until two years ago, when I lost track of him. Usually the death of these people is due to secondary infections which are almost always streptococci.

DR. LOUIS BRUNSTING, Mayo Clinic, Rochester, Minn.: In spite of the absence of edema, I believe that this boy has dermatomyositis. The presenting signs of dermatomyositis may be primarily cutaneous or primarily muscular, and, as our experience increases, we see a number of instances which the muscular phase precedes the cutaneous phase by periods of months or years. In the acute and fulminating forms, edema is quite prominent. About 15% of the cases of dermatomyositis have their onset before the age of 15 years. About one-fourth of the cases fail to live out their second year of the disease. In another one-fourth, the patient is left with a considerable degree of disability from contractural deformities and calcinosis. The outlook in children is usually worse than in adults.

We have used electromyography to help distinguish dermatomyositis from neuropathies, and as a guide in helping to choose the optimal site for biopsy of muscle. The most helpful laboratory test in estimating the degree of activity of the disease is the measure of the serum glutamic oxalacetic transaminase. The sedimentation rate is never elevated to the degree that it is in systemic lupus erythematosus.

DR. S. J. ZAKON: Up to recent years the papers on dermatomyositis in the European literature were published by internists in the various journals of internal medicine. It is of interest to note that in Jadassohn's Handbuch Der Haut and Geschlechtskrankheiten—in all the 42 volumes—the word dermatomyositis is mentioned only once (Vol. IV/1-475). In the textbook "Dermatologie" by J. Darier, A. Civatte, and A. Tzank, published in 1949 (Verlag Hans Huber, Bern. 1949) the word dermatomyositis does not even appear in the index. However, Degos' textbook, "Dermatologie," published in 1953 in Paris, devotes six pages to dermatomyositis.

DR. M. PAUL LAZAR: This past August, Dr. Milton Robin and I had the opportunity of seeing a patient with true dermatomyositis. The total course of the disease was five weeks, and initially the patient had no muscle weakness, which made the diagnosis a little confusing except for color around eyelids and hands. Within five weeks the disease progress was such that she was unable to raise her arms, due to muscle weakness. She had so much edema of the upper extremities that vesicles and bullae were present. In addition, erythema and purpuric-necrotic areas were present over the upper half of the body. In spite of all the inflammation, our patient's sedimentation rate was never elevated. A blood transaminase of well over 500 gave us the clue. The normal sedimentation rate and the very high transaminase would substantiate a diagnosis of dermatomyositis. Within five weeks, despite therapy including steroids, this patient died. Muscle findings, pre- and post mortem, were consistent with dermatomyositis.

DR. ZACHARY FELSHER: Dr. Brunsting, your earliest differentiation was by biopsy of the skin. Does dermatomyositis ever look like lupus erythematosus?

DR. MARCUS R. CARO: In partial answer to Dr. Felsher's question, I have examined a good many sections of the skin, and I cannot recall ever having made a diagnosis of dermatomyositis from the skin biopsy alone. I want to see the muscle biopsy to make a diagnosis, for the skin findings are nonspecific, and at times it is difficult to distinguish between lupus erythematosus and dermatomyositis.

DR. SANFORD: I agree with what has been said. I think we are really all talking about the same disease, and it is just a degree of deciding which one of these myriad of things we have.

I had no intention of giving the impression that I thought antibiotics had any other action except to do away with the secondary invaders. Those are the organisms that cause the mortality in this disease which we find post mortem.

The second point is about the sedimentation rate. In the original description, we can go back and see a description that is very clear. My conception of dermatomyositis is what has been called poikilodermatomyositis, a disease which will conform to the original description. I think the disease today is modified by the use of steroids. What I do want to bring up is the mention of the sedimentation rate. In the original description, it was emphasized that these children had a high sedimentation rate. In this child, the rate is not high, but only slightly elevated. The English do not accept this condition as a disease entity, but say all you have is Still's disease with a rash.

In 1937, Evans presented a paper on poikilodermatomyositis before the British Dermatological Society, and said that the sedimentation rate was always rather high. Whether or not the adrenal steroids have any beneficial effect I do not know. I do not give them to these children. After all, I feel that we are all talking about the same entity. Perhaps my conception of the disease as we know it today has been modified by the adrenosteroids.

In regard to the literature on this disease, one of the best articles ever published on it was written by a member of this Society, Dr. Clark Finnerud in Brennemann's Pediatrics.

Extramammary Paget's Disease of the Perineum. Presented by DR. SAMUEL W. BECKER JR. and (by invitation) DR. JACK M. FOX.

The patient is a 59-year-old white woman, who was first seen at the Research and Educational Hospital in November, 1958. At that time the patient complained of a severe dermatitis in the groin area of approximately 5 years' duration. The patient was apparently well until 1935, when she noted itching of the vulva, and saw a small white lesion with a reddened border on the right labia. She received various treatments, including x-ray. Following the x-ray treatments she developed ulcerations on the labia. These eventually disappeared, but the pruritus persisted. In 1948, she had a simple vulvectomy, with skin transplant, at Billings Hospital. The pathological diagnosis was chronic atrophic vulvitis. The patient was hospitalized again in 1953 because of recurrent atrophic vulvitis, or kraurosis. The pathological report at that time was Paget's disease of the vulva. She had a radical resection done at that time. She remained asymptomatic for a while, but the itching returned. She then developed a flat erythematous scaling area at the site of the previous surgery. This spread to involve the entire groin. She again tried various salves without help. The lesion has become painful, and bleeds easily. Prior to her hospitalization here, she complained of dysuria and incontinence.

Past history reveals the patient to have had two pregnancies. One of these was a miscarriage at two months. Her last normal menstrual period was in 1948. Patient has had numerous operations. These include an appendectomy, varicose vein stripping on left leg, left and right partial oophorectomy, and a partial hysterectomy. Patient also had frequent episodes of diarrhea and abdominal colic. She was told she had colitis in 1953.

The significant findings on physical examination were limited to the groin and perineum. The skin of the perineum was almost completely involved with a beefy, red, ulcerated, well-circumscribed lesion. The ulcerated area bled easily. There was a 1.5 cm. tumor in the left vulvar area. The ulcerative lesions extended 1 cm. into the vagina. The rest of the vagina was soft. The cervix was clean and small. The adnexal areas were soft. Rectovaginal examination was essentially normal. The patient also showed some stress incontinence. There was no apparent inguinal adenopathy.

Laboratory findings showed a normal urinalysis, normal blood count, and a negative serology. The chest x-ray showed slight cardiomegaly. Urine culture revealed some beta hemolytic streptococci, and Staphylococcus aureus coagulase positive.

Biopsies from the nodular growth and the surrounding involved skin are presented.

Histopathological Report.—A biopsy was performed on a typical area of the vulva. Microscopic examination reveals an acanthotic epidermis containing many large, pale, foamy-appearing cells, most of which are seen in small groups. These cells resemble Paget's cells. There is a heavy inflammatory infiltrate just beneath the epidermis. The findings are consistent with the diagnosis of extramammary Paget's disease.

A nodule was removed from the involved area. Histologic sections revealed groups of large, pale, foamy-appearing cells similar to the Pagetoid cells seen in the epidermis.

DR. SAMUEL W. BECKER JR.: I have seen this patient before, but saw the histologic sections for the first time today. I agree with Dr. Caro that these sections look like malignant melanoma. The resemblance between extramammary Paget's disease and melanoma has been described by others. The pathologic diagnosis made at the University of Chicago from the vulvectomy specimen in 1953 was extramammary Paget's disease. There is one test which will differentiate between a malignant melanoma and all other lesions, the tyrosinase reaction.

(Another biopsy specimen was removed later, and the tyrosinase reaction was negative. Although this lesion resembled a malignant melanoma histologically, it was actually extramammary Paget's disease.)

We have recently seen a male patient who presented the same problem in diagnosis. Although he had extramammary Paget's disease of the groin, he developed a malignant melanoma in the same area, and the latter tumor proved fatal.

These tumors which have clear cells in the epidermis present a difficult diagnostic problem.

Juvenile Xanthogranuloma. Presented by DR. ALFRED FALK and DR. S. BIELINSKI, and (by invitation) DR. J. LATONI.

The patient, an eight-month-old baby, first developed skin lesions three months ago on the right frontotemporal area followed by a similar lesion on the internal aspect of the right thigh. Three weeks ago, two new lesions appeared on the right scapular and axillary areas. No subjective symptoms are present.

The lesions consist of red, velvety, nontender, 3-10 mm., isolated, well-limited, indurated papules and nodules, one on each of the described areas. A microscopic slide is presented.

Discussion

DR. LOUIS BRUNSTING, Rochester, Minn.: Recently my colleagues and I (Juvenile Xanthogranuloma with Ocular Involvement: Report of a Case. *J. Pediat.* 54:109-112 [Jan.] 1959) reported on the case of an infant with skin lesions of juvenile xanthogranuloma, in which there was involvement of the globe of the left eye as well. Other cases have been reported, in some of which the eye has been enucleated. In our case, the condition was treated conservatively, inasmuch as the process is considered to be of a benign nature. Over a period of several months, the skin lesions and the condition of the eye have improved. The possibility exists that the lesion may cause blindness, and surgical removal may be required in the future, but the possibility of regression of the lesion, and the development of useful vision, also exists. The case is interesting because the lesions on the skin indicated the probable nature of the tumor of the eyeball.

DR. MARCUS R. CARO: Dr. Senear and I reported a case of nevo-xanthoendothelioma many years ago, in which the lesions disappeared. I have seen a number of cases since then, and, to the best of my knowledge, in all of them the lesions have disappeared spontaneously.

Tonsurous Tinea Capitis (Black Dot Type). presented by MAJOR HENRY L. HINES, Chanute AFB, Ill., and DR. MALCOLM SPENCER, Danville, Ill.

The patient, an 11-year-old white girl, was well until 10 months ago, April, 1958. At that time she began to lose her hair, first in the site of her part, and then gradually to involve the entire crown of the head. She was seen by a local physician, who made a diagnosis of ringworm, and treated with local topical medication without success. Another doctor was consulted, made a diagnosis of alopecia, and local applications of phenol and alcohol were used, and meticortin given orally until a moon face developed, when it was stopped. A basal metabolism rate was reported —30 and —20, and resulted in prolonged thyroid therapy.

Four months ago, some regrowth of hair was noted, but later it fell out again. Six weeks ago all medication was discontinued.

She was first seen at Chanute AFB Hospital December 29, 1958. The entire physical examination was negative except for the scalp. The crown of the head was partially covered by short broken hair remnants. The intact hair was limited to the occipital area. Over the area of fall-out many short hairs were noted, along with fine lanugo type fuzz. The follicles were plugged with black material.

Wood's light examination showed a few scattered spots of equivocal fluorescence. KOH preparation was positive for endothrix, and for ectothrix fungus spores and hyphae. Culture showed a slow growing buff-colored colony. Impression: Tonsurous tinea capitis, black dot type.

Discussion

DR. J. B. HOWELL, Dallas, Texas: This is a very unusual and instructive case. In adults, Trichophyton tonsurans infection of the scalp is a great imitator. After observing this child, it is obvious that great difficulty in clinical diagnosis may occur. Trichophyton tonsurans scalp infections are becoming more common. Epidemics may be anticipated. The family focus is well known. It is well to remove the black dot hairs for cultures.

Adults may acquire this infection from children, and vice versa; as well as having a persistent childhood infection. Apparently, the adult male is not as susceptible to the infection as the female. The clinical features, prognosis, lack of spontaneous cure, infrequency of kerion, and poor response to x-ray epilation are a few of the differences of the scalp infection in adults, as compared to children. Dr. Lewis Pipkin of San Antonio has observed Trichophyton tonsurans keratoderma of the palms and soles in one of his adult patients. I hope the new antibiotic, griseofulvin, will be the answer to this disease.

MAJOR HENRY L. HINES, Chanute Air Force Base, Ill.: I thought twice before bringing a case of ringworm of the scalp to present to a group like this. However, in the short time I have had experience in the Midwest, I have become impressed that this type of ringworm is rare in this part of the country. Fortunately, I was able to study the disease with Drs. Lehman and Pipkin in Texas. The previous case at Chanute I had was a young boy, age 7, who had been under treatment for alopecia areata for one year. What I learned from these cases is that to diagnose this type of ringworm requires a great deal of perserverance. Out of ten cultures, only one showed growth, and out of over 100 KOH samples, only a few were positive. I think the point is that you have to keep trying.

Onset of Psoriasis During Prolonged Course of Steroid Therapy for Asthma. Presented by DR. SAMUEL J. ZAKON and (by invitation) DR. FRED LEVIT.

This 55-year-old ventilation engineer was in good health until 1944, when he was hospitalized for bronchitis. In 1946, a diagnosis of asthma was made, and, while hospitalized for a severe recurrence in 1950, he was given several doses of cortisone.

In 1952, he was again given cortisone, as well as ACTH. From 1952 to the present he has continuously taken steroids, including cortisone, hydrocortisone, prednisone, and at present triamcinolone. The daily dose of steroid has varied from one to six tablets daily. Except for some pimples on his face when he was taking potassium iodide, he has never had any skin disease. In October, 1958, he first noticed scaling patches on the scalp and leg. At that time, he was taking two to four tablets of prednisone daily.

Since about the time of the onset of the skin disorder, he has been taking triamcinolone, one to four tablets daily. He was not aware of any variation in the skin condition when the steroid dose was varied. Since their appearance, the lesions have slowly gotten larger, and the new ones have appeared.

When first seen, the skin lesions had a silvery scale, and Auspitz sign was produced in one lesion. Some improvement was noted on several days' therapy with 5% ammoniated mercury ointment.

Senear-Usher Pemphigus. Presented by DR. DAVID V. OMENS, and (by invitation) DR. HAROLD D. OMENS and DR. CHESTER L. KROLL.

Granuloma Annulare. Presented by DR. ALFRED FALK and (by invitation) DR. RICHARD BERNSTEIN.

Milia Presented by DR. THEODORE CORNBLEET and DR. BERNARD YAFFE, and (by invitation) DR. CHESTER L. KROLL.

Sickle Cell Anemia. Presented by DR. SIDNEY BARSKY and (by invitation) DR. I. GIGLI.

Systemic Lupus Erythematosus. Presented by DR. THEODORE CORNBLEET and DR. BERNARD YAFFE, and (by invitation) DR. I. GIGLI.

Systemic Lupus Erythematosus. Presented by DR. THEODORE CORNBLEET and DR. BERNARD YAFFE, and (by invitation) DR. I. GIGLI.

Secondary Syphilis. Presented by DR. SIDNEY BARSKY and (by invitation) DR. CHESTER L. KROLL.

Alopecia Universalis. Presented by DR. ALFRED FALK and DR. S. BIELINSKI, and (by invitation) DR. R. BERNSTEIN.

Scleroderma. Presented by DR. SAMUEL M. BLUEFARB and DR. SILAS WALLK, and (by invitation) DR. RICHARD BERNSTEIN.

Case for Diagnosis (Chronic Lichenified Dermatitis of the Arms). Presented (by invitation) by DR. WILLIAM GILLESPIE and DR. M. BARRY KIRSCHENBAUM.

Lymphocytic Infiltration of the Skin. Presented by DR. HAROLD SHELLOW.

Case for Diagnosis: Swelling of Face. Presented by DR. JOHN B. HAEBERLIN JR. and (by invitation) DR. B. G. NELSON.

KANSAS CITY-ST. LOUIS DERMATOLOGICAL SOCIETY

Justin L. Mooney, M.D., President
Norman D. Asel, M.D., Secretary

April 5, 1959

Pyoderma Gangrenosa Treated with Electroshock. Presented by JOHN D. DEMOTT, M.D. and WILLIAM H. GOODSON JR., M.D.

The patient, a 33-year-old white woman, was seen in neuropsychiatric consultation at Trinity Hospital on February 4, 1959. Patient presented history of ulcerative colitis of more than twelve years' duration, and recurrent ulcerations of both lower extremities, and other parts of the body, for over five years' duration. The patient had been hospitalized in March, 1956, with ruptured gangrenous appendix, and a resulting fecal fistula of the right lower quadrant. The patient was rehospitalized January 19, 1959, with a diagnosis of chronic ulcerative colitis, fecal fistula, and pyoderma gangrenosa. On admission, patient ran a septic temperature to 102.4, which became normal after about two days. She had a profuse drainage from the fecal fistula, both lower extremities, and presented pyoderma gangrenosa which was severe.

The laboratory findings were white blood count, 15,700: polymorphonuclears, 83%; filamented, 44%; nonfilamented, 39%; eosinophils, 3%. Sedimentation rate 38 mm. in 60 minutes. Urinalysis showed 1+ albumin, and microscopic examination revealed it was loaded with WBC and some RBC.

Medications were as follows:

Jan. 19, Achromycin 250 mg., every 6 hrs., and changed on Jan. 30 to Mysteclin V, 250 mg., every 6 hrs.

Jan. 25, 1959, Corticotropin (ACTH) Jel, U. 45 I.M. o.d.

Jan. 28, 1959, Corticotropin Jel, U. 30 I.M. o.d. On the evening of February 3, the patient became acutely disturbed, psychotic, and delusional, with visual and auditory hallucinations. She was seen in neuropsychiatric consultation, and transferred to Neuropsychiatric Hospital the following day. On the day of the transfer, the patient developed a rectovaginal fistula, with profuse fecal discharge through the vagina.

During hospitalization the patient received 11 EST, Corticotropin Jel, gradually withdrawn, and she was put on Mysteclin V, 250 mg., every 6 hours.

At the time of discharge, February 28, 1959, the patient had undergone a complete remission of all her psychotic symptoms, the rectovaginal fistula

had spontaneously closed, and the spastic colitis had improved to the point that her bowel movements were nearly normal. The fecal fistula was draining very slightly, and the pyoderma gangrenosa had almost completely healed.

The patient has been maintained on electroshock on an outpatient basis, and has continued to improve.

Discussion

DR. RICHARD L. SUTTON JR.: The angle that interests me in this case is the relation between the psychiatric condition and the ulcerated colitis, and sores on the skin and the mental status of the patient. Pyoderma gangrenosa gets better or worse as the ulcers get better or worse. Perhaps Dr. DeMott could say something about the relation of nutrition and psychiatric status. During hospitalization, was this patient on special diet? She got better during hospitalization.

DR. JOHN DEMOTT: Nutritional status is symptomatic of mental condition. She was given no special diet in the hospital. Symptoms change as the patient improves. For example, the patient now has headaches, which she did not have previously. Psychotic patients come in, get psychiatric care in the office, improve, but then develop new symptoms. Many times I have seen this type of patient get worse and worse; then get psychiatric help, and then he gets better.

DR. GORDON SAUER: It would seem from the history that there is a relationship between corticotropin and psychotic symptoms. I think that there is a deficiency in patients with pyoderma gangrenosa. She was given 40 units of corticotropin, and, within 48 hours, showed depression.

DR. MORRIS D. MARCUS: We treat this type of patient with tremendous doses of gamma globulin.

DR. RICHARD L. SUTTON JR.: It is extraordinary to have a psychiatrist among us, and I wish to take advantage of this. I wonder what experience Dr. DeMott has had with patients with other dermatologic problems?

DR. JOHN DEMOTT: I have never administered electroshock therapy for other dermatoses. I have met patients with psychiatric problems who have also had dermatologic problems of the skin, which is a sensitive agent of expression. As a patient improves psychiatrically, his skin improves. The psychiatric patient has oily skin. In my mind I can make a diagnosis of psychiatric need if the skin is oily. Oiliness, flushing, pallor are associated with anger, excitement, etc. In electroshock therapy, the patient who perspires profusely after treatment responds well.

Porokeratosis. Presented by GORDON C. SAUER, M.D.

The patient, a 6-year-old white girl, was first examined on May 28, 1958, at the Dermatology Clinic at the University of Kansas Medical Center. The mother stated that the lesions had begun on the dorsum of the left foot, around the age of 2 or 3. She felt that the lesions appeared first as a crusted area, and then dried to remain as at present. After an episode of chicken-pox in January of 1958, the mother felt that the lesions spread up the leg, and a new one developed on her hand.

In a telephone conversation with the mother on March 17, 1959, she stated that the lesions have spread even further since the child was last seen in November, 1958.

On the dorsum of the left foot, and on one hand, are several light-brownish, flat lesions with a ridge at the border. The lesions do not exceed 2 cm. in size. The center appeared to be atrophic. Some brown, horny, pinpoint-size lesions are present in the center. The child weighed 32 pounds in May of 1958.

A biopsy was done on May 28, 1958, and the report was "hyperkeratotic lesion compatible with porokeratosis."

On June 4, 1958, Kenacort therapy was begun, with a dose of 2 mg. twice a day for four weeks, then 4 mg. twice a day for one week. There was no change in the appearance of the lesion.

Discussion

DR. NORMAN ASEL: This could be a linear nevus, instead of porokeratosis.

DR. RICHARD SUTTON JR.: I should like to see this under the microscope.

DR. GORDON SAUER: I can read the biopsy report. It was seen by the pathologist at St. Luke's Hospital.

DR. NORMAN TOBIAS: I saw my first case of porokeratosis in the Orient. I saw several cases in Japan. They were usually familial. This case looks like those cases I saw in the Orient. It is a nevoid disease.

Lichen Ruber Moniliformis. Presented by BRUCE V. DROWNS, M.D.

A 45-year-old colored woman was first seen April 1, 1958, with a skin condition of three years' duration, which had begun on the backs of the hands, and then spread to involve the arms, chest, back, abdomen, thighs, and neck. There was some itching, but the complaint was mainly cosmetic. The skin was darkly pigmented, and thickened into raised plaques, strands, cords, and islands. Some areas had an orange peel appearance. The cheeks were very firm.

History revealed joint pains since 16 years of age, and irregular heart beat since 1949, for which she takes digitalis from time to time. The patient had a positive serology in the 1940's, and was treated with two courses of penicillin. She states the present eruption began three weeks after receiving treatment for arthritis of the hands with a yellow pill, salicylates, and penicillin.

Serology was positive, urine negative. Her blood pressure was 150/100, pulse 60 and irregular, and her weight was 145 lb.

A biopsy was reported as showing lack of integument, but none of the associated degeneration of collagen with fibroblastic proliferation, such as has been described in lichen ruber moniliformis (the slide is available for examination).

Therapy with Aristocort, 4 mg. three times a day, was started in April, and continued until July 8. At that time, the lesions had become softer, flatter, and less pigmented. Steroids were stopped because of moon face and hair growth on the face. Improvement continued even after steroids were stopped. In January, 1959, she was started on Triquin three times a day for five days, then twice a day, which benefited the arthritis in the fingers.

Multiple Draining Sinus Tracts in Inguinal Regions—Etiology Unknown. Presented by DONALD FUHRMAN, M.D., and LAWRENCE FIELD, M.D.

Erythema Induratum. Presented by GORDON SAUER, M.D.; DAVID MORGAN, M.D., and LAWRENCE FIELD, M.D.

Case for Diagnosis (Bullous Lichen Planus or Benign Mucous Membrane Pemphigus of Mouth and Penis). Presented by GORDON C. SAUER, M.D.

Erythroderma Due to Infiltrative Lymphosarcoma. Presented by LAWRENCE M. FIELD, M.D.

Dissecting Cellulitis—Hidratenitis Suppurativa? Presented by LAWRENCE FIELD, M.D.

Balanitis Xerotica Obliterans. Presented by CHESTER M. LESSENDEN JR., M.D.

Oral Mucous Membrane Pemphigus? Presented by LAWRENCE FIELD, M.D., and FRANK DWYER, M.D.

A Case for Diagnosis. Presented by DAVID B. MORGAN, M.D.

Incontinentia Pigmenti (Reticulated Type)? Presented by DAVID B. MORGAN, M.D.

Tinea Capitis Due to M. Audouini. Presented by GORDON C. SAUER, M.D.

Lupus Erythematosus, Chronic Discoid. Presented by WILLIAM B. O'CONNOR, M.D.

Acrodermatitis Atrophicans. Presented by WILLIAM B. O'CONNOR, M.D.

Pseudo Xanthoma Elasticum. Presented by CHESTER M. LESSENDEN JR., M.D.

LOS ANGELES DERMATOLOGICAL SOCIETY

Dr. Jerome K. Fisher, Secretary

Jan. 14, 1959

Lichen Planus: Paired Comparison Effect of Grenz Therapy and Ultraviolet. Presented by MURRAY C. ZIMMERMAN, M.D.

Pityriasis Rubra Pilaris; Lymphoblastoma? (For Diagnosis). Presented by BEN A. NEWMAN, M.D. and MAXWELL WOLFF, M.D.

Exfoliative Psoriasis with Extravagant Monroe Abscesses. Presented by LYON ROWE, M.D. and (by invitation) SHELDON SWIFT, M.D.

Additional cases presented for discussion:

Dermatitis Herpetiformis (?). Presented by MURRAY C. ZIMMERMAN and (by invitation) FLOYD J. DORNBUSH, M.D.

Atrophoderma Reticulatum. Presented by LYON ROWE, M.D.

A Case for Diagnosis: (Blastomycosis?). Presented by GORDON MACDONALD, M.D.

Case Presented for Diagnosis and Therapeutic Suggestions. Sub-Corneal Pustular Dermatosis; Atypical Dermatitis Herpetiformis; Atypical Lupus Erythematosus? Presented by DR. AYRES and DR. AYRES.

Case for Diagnosis: Possible Mite Bites; Possible Lymphoma? Presented by LAURENCE J. UNDERWOOD, M.D.

Case Presented for Diagnosis and Therapeutic Suggestions? Verruca Vulgaris versus Wart-like Keratoses on Palms and Soles. Presented by DR. AYRES and DR. AYRES.

Case Presented for Diagnosis (Discoid L.E.; Psoriasis of Scalp). Presented by MAXWELL J. WOLFF, M.D.

Submitted for publication March 2, 1959.

MANHATTAN DERMATOLOGICAL SOCIETY

Perry Sachs, M.D., President
Maurice J. Costello, M.D., Secretary

Jan. 13, 1959

A Case for Diagnosis (Folliculitis of Scalp? Seborrheic Eczema? Erythema Multiforme?). Presented by DR. JULES E. VANDOW.

A 27-year-old white man was seen for the first time on January 12, 1959. He is a credit analyst for a bank but spends several nights a week wrestling at a gym. Suddenly, three days ago, an eruption appeared on the scalp, consisting of a palm-sized area of crusting and scaling over the frontal region. Overlying this area are numerous folliculopustular lesions. Inflammatory papules and pustules

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are present throughout the scalp and on the forehead. A half-dollar-sized area of scaling is also present above the left ear. The cervical nodes on the left are enlarged. A group of papules in herpetic arrangement is present over the left side of the chest. A few macules and papules appear to be developing over the chest and abdomen. There is no fever or malaise. No history of drug ingestion can be elicited.

During the week preceding the appearance of the eruption the patient felt tired and had some general muscular aching. For the past six months, because of hair loss, he has been treating his scalp with hair cream, followed by vigorous brushing with a nylon brush. About two weeks ago he began to wear a new hat.

Discussion

Dr. David Bloom: I suggest the diagnosis of herpes zoster on the left arm with generalization of lesions. The profuse eruption on the scalp may be due to locus minoris resistentiae caused by the trauma of the nylon brush.

Dr. Nathan Sobel: This man has generalized herpes simplex. Some of the lesions on the forehead are small pustules with a central dimple, which are seen in Kaposi's varicelliform eruption. This, I think, is a very mild form of that condition.

Dr. Jack Wolf: I agree, but think the history of trauma is important in this case. The brushing has produced a Koebner-like phenomenon on the scalp. In the absence of any trauma from brushing during this early phase of invasion, it is very likely that the scalp would not have suffered this severe reaction.

Dr. M. J. Costello: I think it is generalized herpes simplex. When you see herpes without a preceding dermatosis, it is very unusual. However, the patient had itching of the scalp. Headache, itching, etc. are all symptomatic. When vaccinia is superimposed it is called eczema vaccinatum. You might get a biopsy, and you might inoculate the cornea of a rabbit, or scarify it, to prove the diagnosis.

Dr. J. Harry Katz: I agree with those who believe it is herpes simplex. In the cases I can recall of widespread or generalized herpes simplex, all had a history of frequent "cold sores."

Dr. David Bloom: Herpes zoster may occur without pain.

Dr. A. C. Cipollaro: You don't think it is Kaposi's varicelliform eruption?

Dr. M. J. Costello: That is a misnomer; no one should use the term Kaposi's varicelliform eruption. When Kaposi used it, viruses were not known. I think it should be called eczema herpeticum, or eczema vaccinatum when there is superimposed vaccinia.

Dr. M. J. Costello: I think scabs would not develop at all in herpes simplex, though herpes zoster would cause them.

Dr. A. C. Cipollaro: If there is such a disease as generalized herpes simplex, I have never seen it, and if this is a case, I am glad I have seen it.

Dr. Jules E. Vandow: I saw the man only last night for the first time. He really presented nothing but the scalp lesions, and didn't realize himself that he had any other trouble. The herpetic lesion on the shoulder was not as distinct yesterday as it is today; it appeared following the onset. Most cases of generalized herpes sim-

plex which I have seen have been in children, who have been very ill. The lesions are usually superimposed on a preexisting dermatosis, usually atopic eczema.

Note: Subsequent biopsy revealed a typical lesion of herpes simplex. The patient later learned that his wrestling partner broke out with a vesicular eruption involving the neck and forehead about four days before his own eruption appeared. The distribution was suggestive of herpes simplex.

Acute Parapsoriasis Strikingly Simulating Secondary Syphilis. Presented by DR. MAURICE J. COSTELLO.

Nevus Syringocystadenoma Papilliferum. Presented by DR. JULES E. VANDOW.

Psoriatic Erythroderma Controlled by Corticosteroids. Presented by DR. JULES E. VANDOW.

Pyoderma, Neurotic Excoriations, Acne and Precocious Puberty in an Eight-Year-Old Child. Cushing Syndrome? Mixed Glandular Dysfunction? Presented by DR. JULES E. VANDOW.

A Case for Diagnosis (Verrucae? Neurodermatitis? Keratoderma of Leg and Foot?). Presented by DR. JULES E. VANDOW.

Lymphoma Cutis Presented by DR. JULES E. VANDOW.

A Case for Diagnosis (Lupus Pernio (Hutchinson)? Seborrheic Keratosis?). Presented by DR. JULES E. VANDOW.

Chronic Discoid Lupus Erythematosus: Healing Without Scar Formation in Extremely Severe Case of Twenty Years' Duration, After Treatment with Antimalarial Drugs. Presented by DR. JULES E. VANDOW.

NEW ENGLAND DERMATOLOGICAL SOCIETY

Joseph Goodman, M.D., President
William R. Hill, M.D., Secretary

Jan. 24, 1959

Acrodermatitis Enteropathica, with Familial Incidence of Acrodermatitis Enteropathica and Moniliasis. Presented by DR. WALTER F. LEVER.

A white infant girl, one year old, is presented from the Children's Medical Center, Boston, with an eruption which has been present since the age of 2 months. The rash began around the mouth and chin, and later appeared around the vulva and anus. Its outbreak was associated with diarrhea. It consists of discrete patches, red, oozing, and denuded, with superficial yellow crusts.

Candida albicans was cultured from the lesions. Blood counts and urinalyses were normal. The blood calcium was 9.8 mg. per 100 cc., phosphatase 5.9 mg., sodium and potassium normal. The serum protein was 7.2 gm. per 100 cc., with albumin 5.2 gm., γ_1 5, γ_2 19, β 12, α 13.

The eruption cleared dramatically with the administration of Diodoquin orally, ½ tablet t.i.d. The diarrhea and "fussiness" return if the drug is omitted for 2 or 3 days.

Two brothers died in infancy at the Massachusetts General Hospital, in 1948 and 1955, with a similar rash and diarrhea. One of these also had moniliasis, as do the mother and a sister, whose cases follow.

Moniliasis. Presented by DR. WALTER F. LEVER.

A white woman aged 32 is presented from the Children's Medical Center, Boston, with the complaint of a sore mouth for approximately four years. The patient is the mother of the infant described above, and of the others to be cited.

The patient has complained of a sore mouth, on and off, for many years. When it is untreated, the mouth is red and painful, but there is no exudate. Candida albicans has been cultured on two occasions. The patient felt well while she was taking Diodoquin for six months. She has taken none since before Christmas, and there has been no recurrence to date.

Moniliasis. Presented by DR. WALTER F. LEVER.

A white girl, aged 8, presented from the Children's Medical Center, Boston, is a daughter of the preceding patient. She has had a recurrent slight vaginal discharge for one and one-half years. The discharge is pale white, and the mucosa is reddened and painful, especially on washing. The condition comes and goes without treatment.

Candida albicans has been identified on culture. Laboratory findings are otherwise unremarkable.

Acrodermatitis Enteropathica in Siblings. Presented by DR. WALTER F. LEVER.

Two male infants, brothers and sons of the preceding patients, died in the Massachusetts General Hospital, one in 1948 at the age of four and one-half months, the other in 1955 at four months. Both developed oozing, red patches of skin around the mouth, anus, cheeks, neck, and genitalia at the age of three to four weeks, accompanied by diarrhea. They pursued a fluctuating downhill course until they died of inanition. In both, the initial blood studies and the serum calcium and potassium values were normal. Candida albicans was cultured in the case of the second child, but not in the first. They responded slightly to topical application of antibiotic preparations, gentian violet, and oral and topical administration of Mycostatin. The diagnosis was acrodermatitis enteropathica.

Submitted for publication July 1, 1959.

Discussion

DR. E. WILLIAM ROSENBERG (by invitation): Acrodermatitis enteropathica is a disease in which much is uncertain. Some things are known, however, and these fortunately include the clinical aspects and the therapy. The diagnostic criteria have been well defined since the disease was named by Danbolt in 1942, and reasonably effective treatment has been available since 1953 when the paper by Dillaha, Lorincz, and Aavik was published. (Dillaha, C. J.; Lorincz, A. L., and Aavik, O. R.: Acrodermatitis Enteropathica, *J.A.M.A.* 152:509, 1953.) I would like to concentrate on the useful aspects of what has appeared in the literature, and hope that others will comment on the theoretical considerations.

In most cases there is a familial incidence. The disease almost always begins in infancy, often in the first weeks of life, and usually before the age of 18 months. There is a striking symmetry of the rash, which appears on the face, nape of the neck, buttocks, genitals, elbows, hands, and feet. The distribution is so characteristic that one author has said the illustrations in journal reports are diagnostic. The rash is described as vesicular and bullous, drying and going on to scaling, and as patches of oozing dermatitis with adherent scales at the border. Other manifestations include blepharitis and an intense paronychia with destruction of the nails. There is usually severe or total alopecia. Diarrhea is an important part of the picture; in long-standing cases it may be intermittent. Other findings are a self-centered personality, and a peculiar postural attitude in which the child sits with his head turned partly to one side, with the chin on the chest.

In continued cases, growth is markedly disturbed, and there may be an irritating glossitis. If untreated, the patients with full-blown cases almost always die.

(Slides of the two infants in this family who died are shown.) In these pictures we see the predilection for the areas around the natural openings, and the symmetry of the process. In the case of the second child, Candida were cultured from the skin and stool. This child was treated with nystatin. There was some temporary improvement, then the diarrhea returned, there was wasting and inanition, and the child died after ten weeks in the hospital. The infant presented today had a similar history, and the eruption looked just the same before treatment.

In 1953, Dillaha, Lorincz, and Aavik, at the University of Chicago Clinics, reported successful management of the disease with the use of Diodoquin. The drug had been suggested by Dr. Elias Schlomovitz, who had been impressed by its effectiveness in generalized moniliasis. Since then a number of cases have been reported in which Diodoquin was used. Not all have been successfully treated, and many cases are said to relapse if treatment is stopped.

Diodoquin is the trade name for diiodohydroxyquinolone. Structurally, it is very similar to vioform, differing only in the substitution of an atom of iodine for one of chlorine in the 5 position of the quinoline nucleus. It was first used in the 1930's for the treatment of amebiasis. It is also available mixed with a little boric acid powder and some sugars as Floraquin, and is used by gynecologists in the local treatment of trichomoniasis. It is supposed to be relatively nontoxic when taken by mouth, which probably reflects a low rate of absorption. It is available in 650 mg. tablets.

Some authors have suggested that the entity we call acrodermatitis enteropathica is nothing more than severe systemic moniliasis. Most, however, do not agree, and they point out the strikingly uniform clinical picture, the failure to find Candida in the lungs and other viscera at death, and the apparently well-studied cases in which no Candida were ever isolated.

Another suggestion is that the disease represents an id reaction to intestinal moniliasis. In a paper dating back to 1932, Hopkins reported a similar picture produced with the use of a sterile extract of Candida. Most recently, Danbolt has said he thinks the condition is primarily an intestinal disease with secondary cutaneous changes.

The patients shown today are of special interest, in that here we have a mother and 8-year-old daughter who show proven moniliasis, but no sign of acrodermatitis enteropathica, in a family in which three siblings had the full-blown disease and two died of it.

DR. JOSEPH GOODMAN: Looking at the photographs shown, except for the one depicting what were apparently ruptured bullous lesions, I saw pictures which could readily be taken for patients on whom I make a diagnosis of monilial infection of the skin.

I think there is some confusion in our thinking about monilial infections and seborrheic dermatitis in infants. I suspect that many of the infants on whom we make a diagnosis of seborrheic dermatitis have monilial infections of the skin.

DR. JACOB SWATRZ: I agree with you. I have done microscopic and cultural studies in these infantile cases, and have stated that some of the cases called infantile seborrheic eczema are moniliasis. You get a pure culture of candida. It is not an infection superimposed upon eczema. They are sharply defined, and start out differently.

DR. ALFRED HOLLANDER: I assume the previous speakers are not implying that seborrheic dermatitis and moniliasis are one entity. I hope it is to

be understood that these are two entities to be separated clinically.

DR. BERNARD APPEL: This discussion is significant. I can remember the first case of this entity that I ever saw, presented by Dr. Downing at this society about 20 years ago. The significance of Candida albicans as an obscurer of other clinical signs was impressed on me by that case. Candida albicans as a secondary invader often assumes the predominant role. I feel that the case here presented has individual characteristics indicating that it is not primarily moniliasis.

DR. WALTER F. LEVER: I am interested in the opposing views of Dr. Appel and Dr. Swartz, with Dr. Appel stating that acrodermatitis enteropathica is not basically moniliasis, but that candida organisms are merely secondary invaders, and Dr. Swartz holding the view that acrodermatitis enteropathica is identical with moniliasis.

On the basis of the findings in this family, I am inclined to favor Dr. Swartz's point of view. The two siblings who died, as well as the child presented today, had the clinical appearance of acrodermatitis enteropathica, including the gastrointestinal manifestations, while the mother has oral moniliasis, and an 8-year-old sister vaginal moniliasis. We could obtain Candida albicans from all the areas that we cultured in this child. My view would be that acrodermatitis enteropathica represents a disseminate cutaneous and mucocutaneous moniliasis in specially predisposed individuals.

We have a similar situation, for instance, in the condition called epidermodysplasia verruciformis of Lewandowsky and Lutz. For a long time this was regarded as an entity, until it could be shown conclusively that it represents merely verrucae planae in family members especially predisposed to infection with the virus of verruca.

I believe that in this child Diodoquin has been life-saving. The disease was quite severe when Diodoquin was begun, and even now it has to be given almost continually; if it is discontinued the cutaneous and oral manifestations become active within a few days.

It is interesting to speculate whether we should now start treating patients who have ordinary moniliasis with Diodoquin. I haven't done it yet. I haven't read anything about it in the literature, but believe that a further investigation of the effect of Diodoquin on ordinary moniliasis of the skin is indicated.

Generalized Pigmentation, with Acanthosis Nigricans and Thyroid Nodule. Presented by DR. HOWARD S. YAFFEE and DR. SIDNEY ALLMAN.

A white housewife, aged 41, is presented from Beth Israel Hospital with long-standing obesity, hypertension, and darkening of the skin. The obesity has been present since the age of 12, and the hypertension was first noted eight years ago.

In April, 1958, the patient was seen in the endocrine clinic because of "hoarseness" of two months' duration, associated with a "cold." She said she had been hoarse since childhood, but it had become worse in the preceding five months. There was enlargement of the lower pole of the left lobe of her thyroid, and pigmentation of the buccal cavity, described as blue-black. The blood pressure was 185/115.

She was seen in the Dermatology Clinic on November 6, where the changes of acanthosis nigricans were observed in the body folds. There was darkened brown pigmentation and a velvety appearance of the skin of the neck, where the change occurred first, the inframammary areas, the axillas, groins, and other creases. In addition she has pigmentation of the mouth, palmar creases, in old and fresh scars (e.g., vaccination), and also darkening of nevi. One of these was removed a month ago because of growth, and showed junctional changes.

Studies have revealed no abnormality in the gastrointestinal or genitourinary systems. All roentgenograms have been unremarkable, including skull films, which showed a well-calcified sella turcica without enlargement or erosion. The pineal gland was not calcified.

Thyroid studies showed a radioactive iodine uptake of 25% in 24 hours, with most of the uptake in the lower pole where the enlargement is seen. It was felt that the condition is one of "physiologic overactivity, compensating for thyroid atrophy from prior thyroiditis." The basal metabolic rate was plus 12 per cent.

Of interest in the family history is gout in the father, brother, and uncle. Her father died of arteriosclerotic heart disease and myocardial infarct, and her mother of hypertensive disease. A brother in his 50's has hypertension, while a sister aged 30 is hypotensive.

Laboratory data: 17-ketosteroids were borderline low and normal on two readings; 17-hydroxycorticoids 8.9 mg/24 hr. volume; creatinine 1.87 gm/T.V.; urinary catechol amines were under 180 mg.; serum iron 38 μg/100 cc.; total proteins 7.1, with albumin 4.7, and globulin 2.4; RBC uptake for thyroid function was upper limit of normal, 17%; hgb. 15.6 gm., hematocrit 46%; one-hour PC sugar, normal; thymol, bilirubin serum phosphorus normal; potassium 5.5 mEq.; calcium 11.6; BUN 8 mg/100 cc.

The case is shown to demonstrate local pigmentary abnormality (acanthosis nigricans), clinically, associated with endocrine change and generalized addisonian-like pigmentation; and to differentiate from pseudoacanthosis nigricans,

which may be a metabolic disease, and from localized pseudoacanthosis nigricans, called "Gougerot-Carteaud reticulated and papillomatous dermatosis," among many eponyms, and associated with obesity, clearing with weight reduction. In this syndrome Curtis and Rothman feel that a primary endocrinopathy causes both the obesity and the pigmentation, which may be of the acanthosis nigricans type. However, there is no familial incidence.

Discussion

DR. HOWARD S. YAFFEE: The term acanthosis nigricans today connotes a number of situations which need further clarification. There is little, morphologically or histologically, to distinguish the "benign" from the "malignant" varieties. The benign form is thought to arise before or during puberty, and is not associated with carcinoma. In certain instances it is inherited, as opposed to the malignant form, in which there is a high incidence of carcinoma, both in association with the lesion, and in the families of those afflicted with the dermadrome.

In reviewing the literature, one learns that in the malignant variety the dermatosis may arise on the skin first, with no internal malignancy being detected for as long as six years. Hence, if you encounter a patient in whom the cutaneous changes develop after puberty, there is really nothing to distinguish benign from malignant acanthosis nigricans clinically, in the absence of carcinoma of the viscera.

The really confusing problem occurs in the newly controversial entity of "pseudoacanthosis nigricans" which, strictly interpreted, is a condition arising after puberty, particularly in obese women. The process is usually not as extensive or florid as in the other varieties, and is said to disappear after weight reduction. There are other entities, such as pseudoatrophoderma colli, and confluent and reticulated papillomatosis of Gougerot and Carteaud, which may or may not be associated with obesity, and which may persist after weight is lost. These are regarded by many authorities as variants in the spectrum of pseudoacanthosis nigricans, but there has been great nosologic conflict and recent soul-searching. Basically, this has arisen from the papers of Rothman and Curtis, who have described cases of juvenile acanthosis nigricans associated with endocrinopathy, presumed, but not proven, to be pituitary in origin. Both of the patients described in these reports were obese and "Cushingoid," one aged 23 and the other 42. At postmortem examination, the Chicago patient was found to have pituitary basophilism, despite the absence of definitive chemical abnormalities during life. Authorities vary as to the causative role of pituitary basophilic hyperplasia, since it is found at post mortem in slightly less than a quarter of the cases in some studies.

Dr. Curtis' case, a 23-year-old laborer, was obese, diabetic, and hypogonadic. Adrenal studies were normal. The contention of both groups is that both the obesity and the acanthosis nigricans are of endocrine origin. Kesten, in her paper on pseudoatrophoderma colli, states that H. O. Curth has diagnosed both of these cases as pseudoacanthosis nigricans, rather than that of the juvenile or benign form. It is true that both patients were obese, and photographs did not reveal a florid eruption, but the problem is still difficult to resolve.

Our case is quite fascinating. The patient has a florid, deeply pigmented eruption, with typical verrucous changes. There is conspicuous pigmentation of the mouth, resembling that seen in Addison's disease. This pigmentation has increased since puberty. There is a family history of dark pigmentation, but not of acanthosis nigricans. She has noted a sharp increase in depth of pigment and of verrucous change in the last year. She has been obese since puberty. She also has bitemporal hair recession. Her endocrine studies reveal normal adrenal function, with no suggestion of adrenal atrophy. Her RBC iodine uptake is considered the upper limit of normal, and her radioactive iodine scan shows a functioning thyroid nodule, rather active in the presence of surrounding atrophic gland, presumably rendered so by thyroiditis.

With a functioning thyroid nodule, there is great danger of malignant degeneration. Such a situation, although uncommon, has been reported, and a case of carcinoma of the thyroid associated with acanthosis nigricans has been described. Despite this, from the history, inherited pigmentary anomaly arising at puberty, obesity, long duration, and our frequent vain searches for malignancy, I think this woman has true benign acanthosis nigricans, associated with a primary but undetermined endocrinopathy, possibly associated with or exacerbated by her thyroid status.

It would be most interesting to find that her generalized addisonian-like pigmentation was associated with an increase in melanin-stimulating hormone. Dr. Lerner may have that information for us some day. We have submitted her urine to him for examination. At the moment we do know that her adrenal studies are perfectly normal. Clinically, she lacks a hypoadrenal picture. I think Dr. Lerner has given cortisone to patients with overproduction of MSH, with inhibition of the pituitary as his goal. This has been successful in some cases.

One last point in defense of an endocrinologic concept of benign acanthosis nigricans. If Curth's concept of obesity as the causative factor in

darkly pigmented people is correct, with trauma activating the pigmentation, reversible with weight reduction, it is of interest that the patient of Sheldon and Curtis was obese and blonde. There is much work to be done before the role of the endocrine system in this entity is fully understood.

Acrodermatitis Continua (Hallopeau). Presented by DR. MAURICE M. TOLMAN.

Porokeratosis (Mibelli). Presented by DR. MAURICE M. TOLMAN.

Kaposi's Sarcoma. Presented by DR. MAURICE M. TOLMAN.

Kaposi's Hemorrhagic Sarcoma. Presented by DR. MAURICE M. TOLMAN.

Acrodermatitis Continua. Presented by DR. MAURICE M. TOLMAN.

Psoriasis—Exacerbation Following Triamcinolone Therapy. Presented by DR. MAURICE M. TOLMAN.

Psoriasis—Exacerbation Following Systemic Steroid Therapy. Presented by DR. WILLIAM R. HILL JR.

Exfoliative Psoriasis. Presented by DR. MAURICE M. TOLMAN.

Neurodermatitis (Premycosis Fungoides?). Presented by DR. MAURICE M. TOLMAN.

Sycosis Vulgaris. Presented by DR. MAURICE M. TOLMAN.

Scleredema Adultorum (?). Presented by DR. WILLIAM R. HILL JR.

Erythema Induratum (?). Presented by DR. MAURICE M. TOLMAN.

Cicatrizing Alopecia (Pseudopelade de Brocq). Presented by DR. MAURICE M. TOLMAN.

Poikiloderma Vasculare Atrophicans (Incipient Stage of Mycosis Fungoides?). Presented by DR. PAUL J. CATINELLA.

A Case for Diagnosis (Light Eruption? Early Scleroderma? Lymphocytic Infiltration? Dermatomyositis?—Caused by Contactant). Presented by DR. HOWARD S. YAFFEE.

A Case for Diagnosis (Aberrant (Extrasacral) Persistent Mongolian Spot? Ota's Blue Nevus? Argyrosis?). Presented by DR. FRANCESCO RONCHESE.

A Case for Diagnosis (Granuloma Annulare? Necrobiosis Lipoidica?). Presented by DR. MAURICE M. TOLMAN and (by invitation) DR. SAMUEL MOSCHELLA.

A Case for Diagnosis (Elastoma Intrapapillaris Perforans? Keratosis Follicularis Serpiginosa?). Presented by DR. WILLIAM R. HILL.

Pseudopelade of Brocq. Presented by DR. ARTHUR S. SPANGLER.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILOLOGY

Leslie P. Barker, M.D., Chairman
Nathan Sobel, M.D., Secretary

Nov. 5, 1958

Darier's Disease Resembling Linear Verrucous Epidermal Nevus. Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. LEONARD BROWN.

The patient was a white girl, 25 years old.

History: Patches of verrucous lesions on the right shoulder and right side of the chest, extending over the abdomen, were first noted at about 15 years of age. She saw a dermatologist in Cleveland, who biopsied the lesions on the right side and gave her oral vitamin A tablets for approximately nine months, which gave her no relief. During the next few years, she noted new lesions behind the right ear and within the right ear. She has had almost complete remission of these lesions at times. She noted exacerbations during pregnancy, four times, in 1953, 1954, 1956, and 1957. During these times, she has noted lesions bilaterally on forehead, flexor surfaces of right arm, and axilla. She developed pruritus during past summer. System review reveals evaluation for polyarthritis since 1954 without significant findings.

Laboratory examinations: Pathological report of lesion removed from the back on Oct. 20, 1958: "In these sections of skin from the back examination reveals a verrucous epidermis which is strikingly hyperkeratotic. There are areas of parakeratosis where plugs appear to penetrate into the epidermis. Below these areas one notes lacunae which contain corps ronds and grains. Diagnosis: Darier's disease (keratosis follicularis)." Complete blood cell count, urinalysis, total protein, and A/G ratio are all within normal limits.

Course: No therapy has been instituted.

Discussion

DR. PAUL GROSS: In addition to the nevus-like lesion on the right side of the back, the patient has scattered lesions on the rest of the trunk and typical lesions in the frontal hairline resembling seborrheic dermatitis. Dr. Chester is to be congratulated for demonstrating the fact that Darier's disease, like other dermatoses, may occur in a segmental distribution.

CHAIRMAN LESLIE P. BARKER: Have you ever seen any relationship to pregnancy? This woman said she became worse during pregnancy.

DR. GROSS: I could not say that.

DR. HELEN CURTH: The patient cannot tolerate sunlight well, a complaint which is common in Darier's disease. She also reported that as far as she knows no other member of the family has the disease. Since her parents died very young, when she herself was still a child, and since she does not know anything about her relatives in Denmark or Germany, she may not be correctly informed. Her children are still very young.

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DR. GERALD MACHACEK: We have seen cases with a solitary lesion which was characteristic of Darier's, and I remember a case in which the lesions were unilateral.

DR. BENJAMIN J. CHESTER: I hoped there might be some suggestions in relation to therapy. The patient claims that with each pregnancy these lesions became worse and that she cleared in some areas after each pregnancy. Her so-called "sensitivity to sunlight" is apparently more of a pruritis than an exacerbation of the lesions.

Parapsoriasis en Plaque. Presented by DR. JUAN LARRALDE.

The patient was a 26-year-old white man. The duration of the disease was four and one-half years.

History: The eruption started on the left arm, and new lesions have appeared continuously in the last four and one-half years. No lesions have ever disappeared, once they have developed, according to the patient. Originally asymptomatic, the eruption has become progressively more pruritic in the last year.

Physical examination: On the arms, forearms, front and sides of the trunk, and the thighs are numerous, well-demarcated, erythematous, slightly scaling, flat, nonelevated, noninfiltrated macules and patches of varying size up to 8 cm. in diameter. Many of the lesions on the flanks have an unusual linear appearance. There is also a sharply demarcated, erythematous patch in the groin.

Laboratory findings: Urinalysis normal. Hemoglobin 14.5 gm. WBC 8,600 with 55 polymorphonuclear cells, 23 lymphocytes, 8 monocytes, 9 eosinophils. Total protein 7.2 gm.; albumin 5.6. Sedimentation rate 4 in one hour. VDRL negative. Chest x-ray negative.

Biopsy: "There is a slight hyperkeratosis overlying a well developed granular layer as well as several patches of mild parakeratosis. There is a mild regular acanthosis with an intact basal layer. In several areas one can see a scattering of few lymphocytes in the rete layer. There is a mild perivascular infiltrate in the upper cutis consisting of lymphocytes and some histiocytes lying in a loose, edematous connective tissue stroma about each vessel." The biopsy is nondiagnostic but compatible with parapsoriasis.

Therapy: The patient was started on calciferol (vitamin D_2), 100,000 units daily, on Oct. 28, 1958.

Discussion

DR. PAUL GROSS: There is a combination of lichenoid papules with plaque-like lesions of parapsoriasis, suggesting the diagnosis of parakeratosis variegata. This type of parapsoriasis is usually a manifestation of early mycosis fungoides. Despite the youthful age of this patient, it is important to study his case further with this possibility in mind.

CHAIRMAN LESLIE P. BARKER: This man complains of a great deal of itching. Is that common in parapsoriasis?

DR. GROSS: Yes, in this type.

DR. MAURICE J. COSTELLO: I think he has parapsoriasis. In my experience parapsoriasis does not itch, is not responsive to roentgen rays, and is recalcitrant to most forms of therapy.

DR. MABEL G. SILVERBERG: I think the color of this lesion is very characteristic of parapsoriasis variegata. It is the type which usually ends in mycosis fungoides.

CHAIRMAN BARKER: This patient received x-ray therapy.

Mycosis Fungoides. Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. LEONARD BROWN.

The patient was a 43-year-old Puerto Rican man who has lived in New York for the past 14 years.

History: The patient first noted light-colored spots on the hands and forearms in 1936. These were asymptomatic until 1943, when pruritus was noted. In 1944, hyperpigmentation and thickening of the skin developed and lesions spread to the thighs and gradually to all parts of the body. His first admission to this hospital was in 1948, and numerous hospitalizations followed. Diagnoses ranged from dermatitis medicamentosa, lichen planus-like eruption, heavy-metal intoxication, and parapsoriasis, until 1954, when, after repeated biopsies, a diagnosis of mycosis fungoides was established. Treatment consisted of x-ray therapy to the larger, more pruritic lesions, with regression of the lesions being noted. However, in 1955, the patient developed a large ulcerative, fungating lesion in the left lower scapular area, which was a reticulum-cell lymphosarcoma which responded to mechlorethamine hydrochloride (nitrogen mustard) and radiotherapy. In early 1956, new lesions developed over the trunk and a biopsy specimen taken from a fungating area on the chest was reported as reticulum-cell lymphosarcoma. These lesions responded well to radiotherapy in the order of 2,600-2,800 r. In September, 1956, he was transferred to the Clinical Center, National Institutes of Health, Bethesda, Md., for electron-spray therapy of multiple infiltrative lesions over the body. There was apparent complete regression in all areas treated, but apparently some areas, such as the crural, axillary, and posterior surfaces of the arms, did not receive therapeutic doses, and the lesions persisted in these areas.

Laboratory and x-ray data: A persistent moderate eosinophilia has been noted over the years. A slight elevation of the serum alkaline phosphatase

and thymol turbidity values is a constant finding. The eighth and ninth ribs show pathological fractures, due to radiation necrosis of the bones.

Course: In Sept., 1957, he was again treated with 500 r of conventional x-ray therapy to areas of recurring infiltrating plaques. Areas not treated showed violaceous infiltrative lesions, including the right upper arm, posteriorly; the upper back, and the occiput. Since his last presentation to the New York Acadamy of Medicine, on Nov. 6, 1957, he has shown infiltrative plaques on the face and adjacent to the hyperpigmented areas on the back. Biopsy of the lesion on the back again showed a picture suggestive of mycosis fungoides. He has been treated with emollient creams and 0.1% hydrocortisone to occasional pruritic areas.

Previous presentations to dermatologic societies include the Metropolitan Dermatologic Society, in 1950; the Manhattan Dermatologic Society, in 1950 and 1954, and the New York Academy of Medicine, in January 1954, February, 1956, January 1957, and November 1957.

DR. GERALD F. MACHACEK: Was this the case that originally had the scattering of purpuric lesions?

DR. BENJAMIN J. CHESTER: Yes.

DR. JACK WOLF: I have had occasion to observe this patient on and off for about 18 years. I am impressed with the fact that he looks better tonight than he has on several previous presentations. It simply goes to show what can be accomplished by diligent and assiduous therapy, by keeping after the patient, and by treating lesions as they arise.

For Diagnosis: Mycosis Fungoides? Pruritic Lichenoid Eruption (Generalized). Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. LEONARD BROWN.

Discussion

DR. GERALD MACHACEK: The interesting feature, is that he is worse when he is overheated. We reexamined the slides and found that he had in the material which was available very definite keratosis pilaris in the outlets of the sweat gland ducts, and even though I was aware of the atypical nature of the case, an infiltration of such intensity made me feel I would still have to continue to make a diagnosis of probable mycosis fungoides.

DR. PAUL GROSS: The patient is worse in summertime, and the lesions are most intensive on exposed parts of the body. I would, therefore, consider the possibility of a polymorphic light-sensitivity dermatosis.

DR. FREDERICK REISS: It is very difficult to argue with the histopathologist. I should call it diffuse neurodermatitis, a chronic dermatitis with lichenification due to contact, an undetected allergen.

DR. JACK WOLF: The follicular lichenified appearance of the eruption, its disseminate character, and the absence of any plaque-like suggestion of mycosis fungoides suggest the possibility that the eruption may be neurodermatitis. On occasion neurodermatitis may resemble mycosis fungoides. I do not make any differentiation between neurodermatitis and atopic dermatitis.

DR. JOHN W. DOUGHERTY: I should suggest that the diagnosis of ragweed dermatitis be considered.

DR. ARTHUR B. HYMAN: In view of Dr. Machacek's finding of poral occlusion, I think this might be considered a widespread persistent case of miliaria, with associated itching. Lichenification may be substantial if the poral occlusion goes on year after year, as it probably has in this case.

DR. MAURICE J. COSTELLO: If this eruption represents myocosis fungoides, it is most unusual. It looks something like lichen ruber pilaris, but that disease is rarely seen in the Negro. There is none of the sharp demarcation of the cutaneous lesion with normal skin intervening.

DR. MABEL G. SILVERBERG: I think anybody seeing the slide and not the patient would not hesitate to make a diagnosis of mycosis fungoides.

DR. SILVERBERG: I remember one case of lichenoid mycosis fungoides we had at the Skin and Cancer Unit, but the history was very different. The lesions in that case were reticulated and a little larger than these. A diagnosis of lichen planus had been made on it, but it had a progressive course, not like this course, where the eruption seems to clear in the wintertime and be present in the summer.

DR. BENJAMIN J. CHESTER: I wish to thank the discussers for all their suggestions. I might say that our thoughts were similar to Dr. Hyman's. When we first saw this patient earlier this year we thought we were dealing with a generalized lichen chronicus, secondary to occlusion of the sweat pores, and we did a starch iodine test. This did not show sweat retention. However, examination of the microscopic section showed a striking picture of mycosis fungoides and, therefore, this patient was presented as mycoses fungoides(?).

As for a diagnosis of ragweed dermatitis, this man traveled aboard ship into various areas of the world where ragweed was not present, and he apparently still developed the eruption.

Mycotic Granulomas (Causative Organism, Pityrosporum Ovale?). Presented by DR. HERBERT J. SPOOR.

Location: flexor surface right arm, nape of neck, and surrounding ankles, on knuckles of fingers.

Duration: two months, gradually resolving.

History: a 13-year-old Negro boy, native of Texas, revisited his home during August of this

year. Lesions began as pruritic papules while in Texas and have persisted with gradual resolution until now. Apparently all lesions began at about the same time. Their course consists of moderately painless induration, central necrosis and ulceration followed by gradual resolution with scar formation.

Past history: previous clinic patient at Knickerbocker Hospital: Jan. 17, 1957—acute otitis externa, given S penicillin, 1,000,000 units; Oct. 7, 1958—upper respiratory infection, with CBC, urine, and chest examination within normal range; Aug. 4, 1958—epidermophytosis, mild gynecomastia of right breast, bilateral, mild axillary adenopathy; Aug. 14, 1958—present illness, first visit.

Laboratory: routine chest film, negative. RBC, 4,300,000, WBC, 9,200, with 63% polymorphonuclear cells, 34% lymphocytes, 1% monocytes, 1% eosinophils, no basophils. Routine urine, rare white blood cells, otherwise negative.

Bacteriology and mycology: Pure culture isolated from neck lesion (see subculture specimens).

Colony

A. Nutrient agar (BBH)—(bacteria media): scanty, white, dry, isolated colonies.

B. Sabouraud's agar (fungus media): slow, tan, aggregate, yeast-like colonies.

C. Benham's agar (lipophillic yeasts; Pityrosporum ovale) (oleic acid-wort): tan, moist, smooth; turning darker, dull wrinkled.

D. Pagano-Levin agar (Candida media): No growth.

Morphology

Pleomorphic yeasts with rudimentary endospores similar to Pityrosporum ovales, Type 1 (*A.M.A. Arch. Dermat. & Syph.* 69:323, 1954, and American Type Culture Collection # 7253)

Discussion

Dr. Frederick Reiss: Pityrosporum ovale is a doubtful pathogen. It has as yet not been recognized as the causative organism of seborrhea. If Dr. Spoor can prove that this fungus has caused a granulomatous lesion, this would be the first case in mycological history. I think inoculation with this organism should be tried. If this would produce a similar granulomatous lesion, I think we should accept Dr. Spoor's suggestion that Pityrosporum ovale could produce a granuloma.

Dr. Carl T. Nelson: I doubt that the microorganism isolated is Pityrosporum ovale. P. ovale is a lipophilic yeast and does not grow on simple Sabouraud's agar. There are other saprophytic yeasts on the skin, and it might be fairly easy to recover them from various parts of a little boy like this, who has what seems to be ecthyma.

Chairman Leslie P. Barker: Soon after Dr. Benham had succeeded in isolating and culturing the organism Pityrosporum ovale, I attempted unsuccessfully to produce seborrheic dermatitis by inoculating the cultures on the scalp of children who had been epilated for tinea capitus. I also treated patients with seborrheic dermatitis with a vaccine prepared from cultures of the organism and noted no improvement.

Dr. Herbert J. Spoor: To answer Dr. Reiss, I should not be the first to report a granulomatous lesion from the inoculation of Pityrosporum ovale cultures. This has been reported by others (*J. Invest. Dermat.* 19:289 [Oct.] 1952), although I, myself, have not been able to achieve it in man.

The definition of the P. ovale group varies. At the moment it falls into a Cryptococcus-like group of oil-loving yeasts. It grows very well on Benham's media without dying out. On Sabouraud's it grows for two or three transplants and then dies out. There are numerous reported types, all of which are peculiar yeasts, with rudimentary endospores of some type. I do not think the classification is clear. If the organism grows on an oil-enriched wort agar medium with nutriment supplement as described by Benham and Emmons, then it is a Pityrosporum ovale.

These lesions are not rare. In screening our children for ringworm of the scalp, frequently a flat white fluorescent lesion, such as this, will be seen under Wood's light. If this area is cultured, most frequently one obtains a mixed growth of Staphylococcus pyogenes var. aureus and one of the lipophilic yeasts. In this particular instance I have obtained from an exudative lesion after removing all the surface debris, a pure culture of the particular yeast-like organism shown here.

Dr. Reiss: May I ask Dr. Spoor on what grounds does he classify this as a cryptococcus? To my knowledge that is a yeast-like fungus without filaments and ovale is loaded with filaments.

Dr. Spoor: I cannot classify it as Cryptococcus for the same reason Dr. Reiss gave. However, a classification in one of the medical mycologies recently published in England does classify it as one of the Cryptococcus group.

Scleroderma with Alopecia. Presented by Dr. Thomas N. Graham.

Location: face, ears, scalp, neck, and upper extremities.

Duration: nine years.

History: A white woman, age 65, was first seen by me on Sept. 24, 1958. She complained of an eruption which she stated started on the neck and subsequently spread to involve the above-mentioned areas. The patient has had a heart attack and is also under treatment for glaucoma.

The patient is presented for suggestions as to therapy.

Discussion

DR. MAURICE J. COSTELLO: These lesions suggest lichen sclerosis et atrophicus. There were parallel reticulated, shiny elevations which are characteristic of lichen sclerosis et atrophicus, in addition to the vesicles which she has developed.

DR. PAUL GROSS: I have found the appearance of hemorrhagic bullae pathognomonic for lichen sclerosis. They are the result of mechanical trauma and apparently based on the epidermal changes in lichen sclerosis. One finds these lesions more frequently in cases of lichen sclerosis of the vulva and anus but occasionally on the trunk.

DR. LESLIE P. BARKER: Have you any suggestions for therapy of lichen sclerosis?

DR. GROSS: No.

Multiple Hemorrhagic Hemangioma of Kaposi. Presented by DR. HARRY SHATIN.

The patient was a 27-year-old white (Puerto Rican) man.

Duration: three and one-half years.

Location: extremities, inguinal and axillary nodes, hard palate, hilar area.

History: The patient first developed a lesion on the left sole, which finally ulcerated late in 1956. When first examined in Feb., 1957, in addition to the ulcerative lesion of the left sole there were numerous purplish to brown plaques and papules on the extremities. There were also discrete, enlarged, firm, nontender inguinal, and axillary lymph nodes. A bone x-ray survey and chest x-ray were normal, as were all laboratory studies. Skin and lymph node biopsies were diagnosed as Kaposi's hemorrhagic hemangioma. The skin lesions were treated with x-ray (100 kv., HVL 2.0 mm. Al, 1,200 r in air in divided doses) as were the nodes (1 mv., Thoreus filter, HVL 3.9 mm. lead, 1,500 r in air). There was definite decrease in size of nodes and disappearance of skin lesions with residual pigmentation in a period of several months.

The patient in recent months has developed new lesions on the extremities. These have been treated with superficial radiotherapy as in the past, with definite fading of the plaques to leave residual hyperpigmentation. However, he now presents atelectasis of the right middle lobe visible on x-ray and has developed hepatosplenonomegaly. Also a lesion has developed on the palate in the last week.

Biopsy: The skin shows many new vessels and endothelial-lined blood spaces in the cutis with a moderate amount of hemorrhage. There is an extremely cellular cutis showing a proliferation of a variety of cells from round to spindle shapes. These cells show a few mitoses and variation in nuclear size, shape, and staining.

The lymph node shows foci consisting of many imperfectly formed capillaries, hemorrhage, and proliferating round to spindle-shaped cells.

Discussion

DR. NATHAN SOBEL: I should like to ask why the diagnosis "multiple hemorrhagic hemangioma Kaposi" instead of "multiple sarcoma," which is the usual name.

DR. JACK WOLF: Kaposi's sarcoma usually occurs relatively late in life and runs a course extending over many years before incapacity finally results and before truly sarcomatous spread takes place. In this instance the disease is well developed and rather well advanced in a relatively young man. The prognosis is very bad. By the time he is 40 he probably will have generalized sarcomatosis.

DR. ARTHUR B. HYMAN: I think Dr. Sobel has answered his own question. In view of the fact in many cases the lesions are slow to develop and slow to kill, it may for a long time remain angiomatous rather than sarcomatous. Thus, early lesions of Kaposi may look exactly like benign angiomas for many years.

DR. HARRY SHATIN: I used the term "multiple hemorrhagic hemangioma" since this is the name assigned to this disease in the "Standard Nomenclature of Diseases and Operations" at the present time.

This patient has been presented because of his young age and the very rapid rate at which the disease is developing in contradistinction to the slower development in an older age group as the disease ordinarily presents itself. In addition to the skin lesions there is already involvement of multiple lymph nodes and involvement of the mediastinum, as demonstrated by the atelectasis of the middle lobe of the right lung. He also has an enlarged liver, and, although he has schistosomiasis, which can cause hepatosplenomegaly, Kaposi's sarcoma can involve the liver and may be doing so in this patient.

Hansen's Disease: Lepromatous Type. Presented by DR. ORLANDO CANIZARES.

Familial Benign Chronic Pemphigus. Presented by DR. JUAN LARRALDE.

Pemphigus Benignus. Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. THEODORE A. LABOW.

For Diagnosis: Lichen Chronicus Simplex, Premycotic Stage of Mycosis Fungoides? Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. LEONARD BROWN.

Mycosis Fungoides. Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. THERDORE A. LABOW.

Sarcoidosis and Pulmonary Tuberculosis. Presented by DR. ORLANDO CANIZARES. (Previously presented at the Manhatten Dermatological Society on April 8, 1958.)

Erythema Perstans (Erythema Annulare Centrifugum Type). Presented by DR. BENJAMIN J. CHESTER and (by invitation) DR. LEONARD BROWN.

Metastatic Carcinomatosis (Carcinoma en Cuirasse). Presented by DR. HARRY SHATIN.

Diagnosis: Trichophyton Rubrum, Generalized Five Years. Presented by DR. MARION B. SULZBERGER and DR. RUDOLF L. BAER for DR. NORMAN GOLDFARB.

The patient was a man 27 years old. Sites: toenail, fingernails, dorsum (feet), dorsum (hands), palms, soles, flanks, thighs, buttocks. He has been treated with griseofulvin, 250 mg. q.i.d., since Nov. 29, 1958, and started on 250 mg. b.i.d. since Dec. 18, 1958, because of headache and diarrhea.

Discussion

DR. LESLIE P. BARKER: It might be a good idea to give us just a little résumé about the griseofulvin.

DR. NORMAN GOLDFARB: The drug is an oral antifungal antibiotic, prepared as a white, stable powder. It has been sitting on the shelf, I believe, for the last 20 years. Finally, it was used as antifungal chemical for plants and then in animals with success. Now we are trying it on human beings in the treatment of dermatomyoses at the New York City Skin and Cancer Unit. Dr. Blank has had considerable clinical experience with griseofulvin at the University of Miami. It is especially good in the treatment of superficial fungous infections. Tinea versicolor has proven resistant to date. Griseofulvin is ineffective against the deep mycoses. There is no antibacterial action. Topical application is without therapeutic value. The oral administration has been 1 gm. daily in divided doses. Others have used 2 gm. or as high as 5 gm. daily without any toxic effects. Few side-reactions were noted. This patient had mild transient headaches during the first two weeks of medication. This symptom has not caused him to stop using the drug. In addition, he has had one bout of diarrhea for two days. Resuming the drug has not caused that to return.

DR. LESLIE P. BARKER: Has anyone else had any experience with that drug or have you heard of any toxic effects from it? Have you had more than one case, Dr. Sulzberger?

DR. MARION B. SULZBERGER: I think that this antibiotic, if it lives up to its promise, if no antibiotic-resistant strains of fungi emerge, and if no unknown delayed toxic effects develop, will be one of the very great advances in dermatologic therapy of our day. It is the first antibiotic, indeed the first systemically administered agent of any kind, that really has any beneficial effect upon this tremendous group of diseases caused by the dermatophytic fungi.

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To answer Dr. Barker's question, we have on this antibiotic now about 9 or 10 private patients, and many more in our clinics. I should like to say that the impetus to the clinical use of this antibiotic (which has been on the laboratory shelves since 1939, when it was isolated from several species of penicillium by the original group of H. Oxford, Raistrick, and co-workers), came from the investigations of Gentles, in Glasgow. Gentles reported in *Nature*, in late 1958, that griseofulvin, administered by mouth to guinea pigs, infected with Microsporum and Trichophyton fungi, was both effective and nontoxic. Other investigators then also demonstrated the lack of toxicity of griseofulvin in rats, unless huge doses were injected—many multiples of the therapeutic doses. These results encouraged several dermatologists to use it in man. The first to use it in man were, as far as I can ascertain, Dr. Peterkin, Dr. Calnan (who is here tonight), and several others. But these investigators applied it only locally and found that it had no therapeutic effect when put on dermatophytic infections as topical applications, even in high concentrations (5%). Dr. Harvey Blank and Dr. Roth, in this country, and Dr. Williams and co-workers, in England, were the first to report on the clinical value of griseofulvin when given by mouth to patients with various kinds of dermatophytic superficial fungous infections of the skin, hair, and nails. I believe Dr. Pardo-Castello in Cuba and Dr. Riehl Jr., also were among the first to observe its systemic effectiveness. At that same time, Dr. Frederick Reiss and Dr. Rudolf Baer of our group, having heard of the work of Gentles (Dr. Reiss while attending the International Congress of Mycology in Lisbon, and Dr. Baer through having been in prior correspondence with Dr. Gentles and having read his article in *Nature*) quite independently wrote to the Glaxo Laboratories in Middlesex for some of the griseofulvin. Dr. Blank also at about the same time received the drug from this laboratory and treated about 30 cases with the encouraging results that Dr. Goldfarb has mentioned.

It is interesting to learn that as far as medicine is concerned, this potentially valuable drug for man was just put on the shelf after its discovery, in 1939, and that it was then first used to treat fungous diseases of lettuce and tomatoes, then of cattle, and now, finally, after 20 years, at last applied to diseases of man.

Of course there are exciting possibilities here for further study. Griseofulvin is a relatively simple chemical with no nitrogen in its structure but only carbon, oxygen, hydrogen, and chlorine.

The possibility that by modification structures of this kind may prove useful against other fungi, against other types of bacteria and micro-organisms, and even against virus, perhaps, is very challenging. Many problems concerning griseofulvin itself must still be solved. Whether one will have to treat these patients with Trichophyton rubrum infections for many years in order to clear them definitively is not known, but if our theories are correct, then patients with active T. rubrum infections of the skin and of the nails certainly are different from other people in their susceptibility to this infection. You know, of course, that very often in the families of such patients one finds other persons with infections. Dr. Martin Fisher of our group and other people have shown that when one member of the family has active T. rubrum infection, one can by careful examination of all the others living with them often demonstrate that some have almost inapparent foci which they do not recognize as being disease. In these one can demonstrate the fungi in a callus, in a scaly area between the toes, in the toenail, or elsewhere, but these other family members virtually never get any widespread disease which is active and manifest. So the actual patients with active widespread fungous disease, once they are "cured" by this antibiotic clinically, will still often be going back to the foci in their homes and families. Moreover, many apparently "cured" patients will still be having inapparent fungus colonies living in their own nails or elsewhere. So unless their immunity to the fungus has been raised, by the griseofulvin or otherwise, the probability is that many will come back with either autogenous or exogenous recurrences of active disease some time after the antibiotic has been stopped and eliminated.

I could go on for a long time, but I have spoken too long already. However, there are many other interesting things about this particular antibiotic, not the least of which is that it has two entirely new effects. It does not seem to kill the fungi but just seems to act in the tissues, in the keratin, and upon the surface of the fungi as a fungistatic agent. Roth and Blank reported that when they washed off the griseofulvin from the fungi which had been stopped in their growth by being immersed in the relatively dilute solution, the washed fungi go right on living again as though they had not been exposed to the antibiotic. Moreover, again according to Roth and Blank, patients that are receiving therapeutic doses of griseofulvin by mouth do not have any significant antifungal activity in their blood serum. Apparently the griseofulvin does not begin to act as a fungistatic until it gets out of the blood and into the tissues or keratin where the fungi are. It seems to me that the simplest hypothesis is that there is something in the circulating human blood that inhibits the fungistatic action. Another very interesting effect requiring further study is that in huge doses given to animals by injection (many multiples of the therapeutic dose) griseofulvin has a colchicine-like effect in arresting mitoses in the metaphase.

DR. FREDERICK REISS: I just have one short remark, that this antibiotic has been assayed in the skin and also in the blood, and, as Dr. Sulzberger emphasized, the blood and skin levels are hardly appreciable. At present we have under our treatment about 25 cases at Bellevue Hospital; I presume in one to two weeks we may see results from this drug.

In relation to Dr. Goldfarb's case, there are two interesting findings, apart from what he has already said, in relation to the disappearance of itching and scaliness of the diseased skin. There is something which I presume everybody noticed, that below the lunula the nail already shows the sign of a healthy growth. In addition to that, it is noteworthy that on the palm where perspiration was absent before the treatment it was freely perspiring after griseofulvin medication. I noticed this phenomenon already in China, in Trichophyton purpurum infections of the palm, a striking symptom; without making a thorough mycotic investigation one could surmise the possibility of a T. purpureum infection on the basis of the absence of perspiration and the nail changes.

DR. MAURICE J. COSTELLO: Dr. Goldfarb referred to the fact that the moniliasis is made worse by the administration of this drug. That should be kept in mind since such candida infections become systemic. I think our greatest problem is not the fungus infections of the glabrous skin but of the nails. If this drug cures fungus infections of the nails, I would be perfectly happy about it, because the type of fungus infection that resembles dermatitis herpetiformis and others responds to local therapy, even though the patient has had the disease for many years.

DR. RUDOLF L. BAER: The recovery of this patient's ability to perspire is most interesting. Some years ago Dr. Muskatblit and I reported on a patient who had a generalized T. rubrum infection superimposed on a congenital dermatosis which had been diagnosed as erythroderma of the ichthyosoform congenital type. We studied this patient thoroughly mycologically, and, with the help of Dr. Herrmann, a certain number of physiologic studies were also done. After the patient had been successfully treated topically for his fungous infection, he noted that he could perspire all over the body and on the palms and soles for the first time since he could remember.

DR. DAVID BLOOM: What is the effect of the drug on tinea capitis?

DR. BAER: I have no personal experience with treatment of tinea capitis with this drug, but I have been told that Dr. Blank treated several cases of tinea capitis with success.

DR. JESSE A. TOLMACH: I should like to know how long it took for the first evidence of improvement to appear.

DR. NORMAN GOLDFARB: As concerns the glabrous skin, it takes about five to seven days before the itching stops and the erythema and scaling is decreased. On the nail, because of its slow growth, one does not notice improvement until about two or two and one-half months have passed. You may be disappointed, therefore, if you look at the nails expecting progress in a shorter time. But as the nail grows out there is a definite demarcation between the white opaque portion involved with T. rubrum and the new portion that is growing healthy and pink, as a nail should be. This line of demarcation keeps progressing outwardly even after medication has been discontinued. You cannot see it in the examination of this patient tonight. Probably in another month you will be able to detect this phenomenon peculiar for this drug.

For tinea capitis, three weeks' treatment at least is required with griseofulvin. The program used at present calls for 1 gm. daily. After three weeks you may shave off the hair of the scalp to remove the infected hairs.

Localized Myxedema; L.E.? Presented by DR. A. I. WEIDMAN.

History: The patient, a white woman, age 44, has had the present lesion on the chin for the past five years. The lesion began as a "small red spot", generally became larger until it reached its present size, about two years ago. There was no history of injury with glass. The biopsy reports performed elsewhere were not conclusive. Her physician gave her Rimifon, Aralen, vitamin D, injections of streptomycin, "tranquilizers," and various local medications. At the Mt. Sinai Hospital the patient received one injection of streptomycin *into* the lesion, injections of penicillin, and, orally, Calciderol, INH, chloroquine. In October, 1957, the patient was seen at New York Hospital where a third biopsy was taken and the patient was given Rimifon. In September, 1958, the patient came to the Skin & Cancer Unit, where the fourth biopsy was performed.

Physical examination: At the right side of the chin the patient exhibits a violaceous, diffuse, slightly raised, and moderately infiltrated lesion. A few biopsy scars and superficial wrinkles are noticed on the surface of the lesion. The regional lymph nodes are not palpable.

Work-up: CBS, essentially normal; STS, battery of quantitative tests, negative; urinalysis, negative; BMR, Nov. 13, 1958, —16%.

Biopsies: at Mt. Sinai Hospital on Nov. 11, 1954: "Lupus Vulgaris type of reaction." At New York Hospital: "Lupus Vulgaris reaction." At the Skin & Cancer Clinic (Dr. Miller): "Granulomatous reaction with a lupus vulgaris type of response."

Dr. Rothstein and Dr. Hyman, tuberculid of the face.

Dr. Sims, Dr. Pensky, and Dr. Morell, tubercle formation as may be seen in sarcoid or tuberculosis of the skin.

Dr. Richter: "Granulomata strongly suggestive of Boeck's sarcoid but the possibility of an associated condition causing the lymphocytic infiltration should be considered."

Dr. Pincus, Monroe, Mich: "No sarcoidosis granuloma faciale, L.E. lymphocytic infiltration, or alopecia mucinosa. The pronounced edema on the face makes one wonder whether there might be mucinous substances in the corium, but localized myxedema of the face certainly would be most unusual."

O.T.: intracutaneous tests (right forearm). (1) 1:1,000,000, neg.; 1:100,000, neg.; 1.1,000, neg.

Roentgen and dental examination of the lower jaw: "Essentially Negative."

Roentgenogram of chest (Dec. 18, 1958): "Examination of the chest shows left ventricular enlargement with the transverse cardiac diameter being fully 1 cm. above normal. There is no recent parenchymal lesion or pleural abnormality. There is a moderate dorsal kyphyosis with moderate osteoarthritis."

Present therapy: Kenacort, 4 mg. t.i.d. for about four weeks; no response as yet. Proloid, ¼ grain (0.016 gm.) t.i.d.

Discussion

DR. DAVID BLOOM: If no definite diagnosis can be made, I would suggest the diagnosis of lupus tumidus.

DR. JACK WOLF: On the basis of several—and I think there were several—histologic examinations suggesting lupus vulgaris, I am inclined to go along with the diagnosis that Dr. Bloom has rendered.

DR. MAURICE J. COSTELLO: I have seen this patient before at one of the meetings. At that time I suggested lupus pernio. It is not unlike those lesions we see around the nose and mouth. It does not respond to therapy; it changes very little, and a tuberculoid reaction is present in the histological examination.

A Case for Diagnosis: Facial Granuloma with Eosinophils (Pinkus) Presented by DR. JESSE A. TOLMACH.

History: The patient, a 22-year-old woman, a dental technician, has had an eruption limited to

the face for the past three years. Exacerbation of lesions has occurred in cold weather. There was no clearing of eruption for longer than one month in the past year. The general health has been good. There is no history of internal medication and no response to therapy with acne lotions, topical hydrocortisone, three x-ray exposures to face.

Physical examination: Lesions consists of grouped papules on an erythematous base. There is a suggestion of annular configuration. No atrophy.

Laboratory findings: Biopsy performed by outside doctor, May 17, 1958, revealed the following findings: "The epidermis is somewhat acanthotic. The upper third of the cutis is occupied by a granulomatous reaction. This is perivascular and in the capillary bodies. The reaction is composed of round cells, wandering connective tissue cells, and leukocytes, with a predominance of eosinophils. Many histiocytes are also present. Eosinophilic granuloma of face."

Results of a biopsy done at the Skin & Cancer Unit on Oct. 8, 1958, were as follows: "A relatively normal epidermis. The uppermost cutis is edematous. The vessels are dilated. The upper one-fourth of the cutis shows aggregates of inflammatory infiltrate around the vessels as well as more scattered elements, composed of small round cells, PMNs, and occasional eosinophils. The basal margin of the surface epidermis as well as of the follicles is intact. It is probable that this belongs to the group of 'facial granuloma with eosinophils (Pinkus).'"

Complete blood cell count Nov. 12, 1958: hemoglobin 12.6, red blood cells 4,250,000, white blood cells 7,800: polymorphonuclear leukocytes, 56%, lymphocytes, 31%, monocytes, 1%, eosinophils 12%.

Discussion

DR. ARTHUR B. HYMAN: I examined the slides at the Skin and Cancer Unit. They show the infiltrates in the skin described by Pinkus in facial granuloma with eosinophils, which is a condition distinct from eosinophilic granuloma. The difference is that the eosinophilic granuloma is part of the set of complexes now known as histiocytosis X, whereas the facial granuloma with the eosinophils is some kind of infiltrate in the skin (usually of the face) that is not related to Hand-Schüller-Christian disease, eosinophilic disease of bone or Letterer-Siwe disease. There are histologic differences between the two conditions which I do not think it would be fair to go into without showing them under the microscope.

DR. JESSE A. TOLMACH: As Dr. Hyman has said, this is a distinct histological entity. Apparently, it is not a distinct clinical entity, because the description in this case varies from the description of other reported cases. It is interesting to note that the lesions did not respond to superficial x-rays, CO_2 snow, topical corticosteroids, or lotions containing sulfur and resorcin. Some of the lesions tend to subside spontaneously after a few months, but new ones continue to appear.

Nevus of Ota. Presented by DR. ALFRED W. KOPF.

The patient, a 9-year-old Negro girl, was born with a bluish discoloration involving the left half of the face, including the forehead, temple, and zygomatic and preauricular areas, as well as the palpebral and scleral conjunctiva. According to the history the child was born with the present condition, and it has gradually lightened over the years. The patient has been of normal development otherwise and has kept up with her contemporaries in school. Neurologic and ophthalmologic examinations have not revealed any abnormal findings.

Examination: At the sites noted the patient presents a macular, bluish-black discoloration which is ill defined on the cutaneous surface where the areas of hyperpigmentation gradually merge with surrounding normal dark skin pigmentation. The sclera of the left eye (or the conjunctiva overlying the sclera) is studded with various irregular blotches of dark blue pigmentation.

Histological findings (biopsy of skin of left temple) were reported as follows (Dr. Charles Miller): "The configuration of the epidermis is normal. There is marked pigmentation in the basal cell layer. Thinly scattered throughout the corium one sees round asteroid and ribbon shaped melanocytes. Nevus of Ota."

Discussion

DR. RUDOLF BAER: This is certainly an unusual case, but apparently in some other parts of the world this type of nevus is not as unusual. Dr. Kaminsky, of Buenos Aires, published a series of seven such lesions in Caucasians which he observed within a period of two months. In his paper he stated that among the yellow races this type of nevus is observed once among 200 dermatological patients. This nevus had been supposed to be exceedingly rare or practically nonexistent in Caucasians; apparently the incidence of it is higher but still very small in the Negro race. Nevus of Ota occurs predominantly in women and involves not only parts of the eye itself and the skin but in some cases pigmentation also is found in the eye muscles, in the periosteum and in the retro-orbital fat.

DR. GERALD F. MACHACEK: I was a little forewarned about this thing, so I attempted to get some information about it. As far as I can make out, Dick Elder does not mention it. In the very fine recent book, Dr. Reiss does not mention it. It is a type of nevus which ophthalmologists consider to be derived from the so-called mesodermal

melanoblasts. It is probably a form of blue nevus of the cornea, although they mention blue nevi of the eyelids. I believe the ophthalmologists do not recognize the conception we have, that melanoblasts arise from the so-called neural crest, but feel that this type of nevus is neurogenic. They also discuss in their fundamental discussion of melanosis the mesodermal and epithelial. However, it seems to me that this probably fits in with the category of blue nevus of Ito and Yoshida.

DR. HELEN CURTH: This is a blue nevus. I believe the prognosis in this case is good because cases in which melanomas ensued were those in which, as Dr. Baer pointed out, the deep tissues were involved. This does not seem to be the case in the patient tonight. The patient also reports that the discoloration has been getting lighter. A good description of this condition can be found in R. L. Sutton's "Diseases of the Skin," St. Louis, C. V. Mosby Company, 1946.

DR. ARTHUR B. HYMAN: Dr. Machacek just spoke about the origin of the melanin-forming cell. He started off by suggesting a mesoderm melanocyte. I think most embryologists and anatomists who have studied it, and the dermatologists who have followed in their footsteps, have come to the conclusion that the pigment-forming cell, that is, the melanocyte, is always neuroectodermal in origin, and this is true also of the cell of the blue nevus, the Mongolian spot, and Ota nevus, which are essentially the same. The presence of pigment in the muscular or periosteal tissue is not to be unexpected. Wherever melanin is formed rapidly or in large quantities some of it is likely to get out of the melanocytes and reach the tissues around. This is often absorbed by the reticuloendothelial cells, which then are called melanophages. Just by looking at a cell it may be difficult to distinguish between a melanophage and a melanocyte. The pigment, found in this case in the deep structures, the muscle and periosteum, is probably present in cells that have been absorbed but not formed it themselves.

DR. LESLIE P. BARKER: Could you tell us if any of these cases have ever developed into melanoma?

DR. ALFRED W. KOPF: Dr. Ota described this condition under the title of "Nevus Fusco-Caeruleus Ophthalmo-Maxillaris" (*Tokyo Med. S.* 63:1243-1245, 1939). The largest series of patients with Ota's Nevus were reported by Ito and Yoshida (*Tohoku J. Exper. Med.* (Supp. 1) 55:34-43, 1952) in which they reviewed 110 patients with this entity. Ota's Nevus is present at birth in 60% of the cases, and, as Dr. Baer said, affects predominantly females. It is a bluish discoloration which involves most often the skin innervated by the first and second division of the trigeminal nerve. The lesion may fade (wholly or partly) over a period of years.

In answer to Dr. Barker's question, to my knowledge there have been only two cases reported that have developed malignant melanoma within such nevi. These patients were reported by Drs. Dorsey and Montgomery (*J. Invest. Dermat.* 22:225-236, 1954).

In Japan, where this is apparently a relatively common skin condition, it is usually not seen in the light-colored Japanese people but affects primarily the brunette. One can think of Ota's Nevus as a misplaced Mongolian spot which has a lesser tendency to disappear.

Drs. Ito and Yoshida demonstrated that the cells responsible for the condition are stellate dopa-positive melanocytes.

Intraepidermic Tumor. Presented by DR. SIDNEY ROBBINS, DR. CHARLES SIMS, and DR. FRANK KRASNER (by invitation).

History: The patient, a 51-year-old woman, noted a lesion in the location of the present one many years ago. It appeared as a "wart" to her and was "burnt off" by a doctor. Several years later a "discoloration" was noted followed by scaling which periodically came off. One year ago the nature of the scales apparently changed in that the latter became firmly attached. In July, 1958, the scales were removed by another doctor and the area remained "raw" for a long period thereafter.

Description: Over the lower part of the abdomen is a quarter-sized, sharply demarcated plaque level with the surface of the skin in some areas and raised in others. The surface is covered by an adherent seborrheic type scale.

Microscopic examination (Dr. Charles Sims): "The epidermis is moderately and irregularly acanthotic. There are numerous intraepidermal clusters of cells which are fairly uniform and reveal a poorly staining cytoplasm and a round or oval vesicular nucleus. Many of the cells have separated from one another, resulting in lacunae formation. Occasional mitoses are present. Most of the above process is suprabasilar. Occasional individual cells with pale staining cytoplasm are seen in the upper layers of the rete. The upper corium presents dilated vessels surrounded by a moderate cell reaction composed of lymphocytes, histiocytes and plasma cells. Edema of the perivascular connective tissue is evident."

Discussion

DR. FRANK KRASNER: The reason the diagnosis was put as such is because the patient was very worried about the lesion and we decided not to put down a definite diagnosis. The slide is the most interesting factor in this case. It has a very

peculiar type of cell separated from the surrounding cells and lying freely in vacuolated areas in the epidermis. It has been shown to several histopathologists. Dr. Richter of the University Hospital (New York) felt it was an intraepithelial malignancy but did not specify the type. Dr. Hamilton Montgomery, of the Mayo Clinic, felt that it was probably extramammary Paget's disease. However, he stated that had the slide been shown to Dr. Arthur Allen, the latter probably would have said it was a superficial melanocarcinoma. The patient was shown at a recent meeting of our Tumor Conference at Skin and Cancer (New York), and it was felt this might be a Bowen's disease.

DR. ALFRED W. KOPF: I think there is an additional point in the history that should be recorded. This woman states that she had taken drops for acne over a period of several years and had the prescription repeatedly filled. Can one assume that this was arsenic?

DR. ARTHUR B. HYMAN: Clinically one would have said this is a basal superficial cell epithelioma or Bowen's disease. The diagnosis is based on these histologic features: there are whole groups of cells which, to a certain extent, are separated from their neighbors. The cells are sort of vacuolated. The sharp grouping is reminiscent of two conditions, Paget's disease and malignant melanoma. I do not think that this is an example of either of those two diseases. There are also present in the epidermis, between these particularly sharp groupings of cells, the features of Bowen's disease. Histologically this is a subvariety of Bowen's disease, and therefore I would call it an intraepidermal epithelioma.

DR. SIDNEY ROBBINS: In conclusion, I should like to state that Allen and Spitz have suggested a more all-inclusive name to this entity; namely, "pagetoid melanocarcinoma." These authors do not accept the concept of extramammary Paget's disease, as no apocrine or sweat glands are seen in this type of lesion.

Melanocarcinoma. Presented by DR. O. CANIZARES and DR. A. CASEY (by invitation).

The patient, a 76-year-old white man, states that three months prior to his first visit to the skin clinic of Bellevue Hospital, he noticed a small, erythematous elevated lesion in the right preauricular region. Since he had a similar lesion in the left preauricular region of three and one-half years' duration, he paid no attention to the lesion in question. However, approximately one month preceding his visit, the right preauricular lesion commenced getting larger and reached the present size of 1×1.5 cm. A punch biopsy was performed and it revealed a melanocarcinoma. He was then admitted to the Service of Dermatology of Bellevue Hospital, at which time the biopsied area had healed and blended into the texture of the lesion which was an irregularly outlined, indurated and erythematous patch without pigmentary changes. However, on Jan. 2, 1959, melanin pigmentation was first observed, and as of now it is readily apparent.

The chest x-ray was not remarkable except for cardiac enlargement.

Ophthalmologic examination failed to reveal any involvement.

The Fourth Surgical Division contemplates doing a total excision of the lesion with a free border of at least 1 cm. No gland dissection is planned because of moderately advanced cardiovascular disease.

Discussion

DR. EDWARD H. MANDEL: Mr. Chairman, I think the protocol states, "Wide local excision is contemplated." I should like to suggest, on the basis of statistics (Lane, N.; Lattes, R., and Malm, J.: The Surgical Pathology of Malignant Melanoma of the Skin of Adults: A Clinico-Pathological Study of 117 Cases, *Bull. New York Acad. Med.* 34:824-827 [Dec.] 1958), that regional node dissection be carried out in addition to this. This is especially valuable when the regional nodes are not palpable, since occult metastases can be detected and the patient's prognosis can be improved.

DR. LESLIE P. BARKER: How many nodes would you suggest? Where would you stop?

DR. EDWARD H. MANDEL: I am not a surgeon.

DR. LESLIE P. BARKER: This is the trouble. When they get started on these they don't know where to stop.

DR. RUDOLF L. BAER: In the history it was stated that this patient neglected the lesion in front of the right ear, because he had had a somewhat similar lesion in front of the left ear. Indeed examination reveals that he does have an erythematous elevated lesion there. It would be interesting to know the nature of this smaller lesion.

DR. ORLANDO CANIZARES: The interesting point in this case was that the original lesion did not at all resemble melanoma. It was a pale lesion and it was thought to be possibly a sebaceous cyst, and the possibility of a basal-cell carcinoma was considered. Therefore, a simple excision of the lesion was performed. To our surprise, it turned out to be an amelanotic melanoma and the lesion relapsed. This is the lesion you see at present, which has a dark spot in the center. As far as the treatment is concerned, the surgical department has decided to carry out a simple excision with a wide margin without removal of the lymph nodes, especially since the patient is 76

years old and has serious vascular disease. It was felt the least surgical trauma in this case would be best.

Linear Scleroderma. Presented by DR. ARTHUR B. HYMAN and DR. JAMES ROCHFORD (by invitation).

History: The patient, a 6-year-old white girl, 15 months ago developed suddenly a reddish streaked lesion 1 in. wide, starting on the right shoulder and running down to the right thumb. Twelve months ago it was described as a discrete confluent mainly purplish-red band of irregular outline from shoulder to the thumb. The distribution of the lesion was mainly on the medial flexural aspect of the arm.

X-ray examination revealed a small cervical rib on the right side but not the left, and also spina bifida occulta involving the 12th vertebra. Vascular investigation showed no abnormality, and the patient had no symptoms referrable to the cervical rib.

Six months ago whitish areas were noted in the previously involved areas. There was irregular indistinct border which appeared slightly violaceous in certain areas, with some areas showing hyperkeratosis. Local therapy of 3% LCD for three months and hydrocortisone ointment and Aralen (100 mg. daily) for three weeks showed no improvement.

Physical examination: At present patient shows on the right shoulder and deltoid region, circumscribed partly isolated, partly confluent whitish flat lesions. Surrounding the lesion is a hyperpigmented zone. Over the deltoid area there are completely whitish, atrophic plaques which have a definite feel of superficial hardening. The normal skin architecture is absent. The lesion follows a course from the right shoulder over the medial aspect of the arm and the flexural surface of the elbow, forearm and wrist. On the radial aspect of the right wrist are several punctiform deep bluish angiomatous lesions. On the thenar aspect the blood vessels show a reticulated network.

Microscopic examination: Jan. 15, 1958, University Hospital: There is moderate hyperkeratosis. The papillary layer of the corium is hyalinized and edematous. All layers of the dermis are diffusely infiltrated by a moderate number of lymphocytes and occasional leukocytes.

Diagnosis: Scleroderma.

Discussion

DR. MARION SULZBERGER: This is the case of the little girl with linear scleroderma. This evening we have seen so many patients who cannot be helped that I am glad to think that perhaps we can help this child. She happens to have a cervical rib and the scleroderma has developed on the same side as the cervical rib. She also has a spina bifida occulta. I wonder if removal of that cervical rib might not relieve the scleroderma.

As we know, linear sclerodermas often appear associated with osseous changes which affect the nerves and nutrition, first probably compressing or irritating the nerve and then producing trophic changes in the areas served by that nerve. I wonder if this cervical rib could not be removed and if that might not help.

CHAIRMAN LESLIE P. BARKER: Has anyone any suggestion? Has anyone any experience with the removal of cervical rib? There seems to be nothing but silence.

Could the presenter report if there is any work on it?

Lymphoblastoma Type (1) Mycosis Fungoides, (2) Hodgkins? Presented by DR. J. H. POLLOCK.

Unusual Multiple Subepidermal Nodular Fibroses (Noduli Cutanei); Dermatofibrosarcoma; Kaposi's Sarcoma; Keloids? Presented by DR. ARTHUR B. HYMAN and DR. ALVIN M. LASHINSKY (by invitation).

Chronic Bullous Erythema Multiforme (Pemphigoid?); Pemphigus Vulgaris; Dermatitis Herpetiformis? Presented by DR. MAURICE J. COSTELLO and DR. B. GORDON (by invitation).

Pemphigus Vulgaris. Presented by DR. ORLANDO CANIZARES and DR. RICHARD CARL GIBBS (by invitation).

Tuberculosis Verrucosa Cutis of Leg and Buttocks. Presented by DR. O. CANIZARES and DR. R. STEAGALL (by invitation).

Hypertrophic Lichen Planus. Presented by DR. S. BLAU and DR. N. KOS (by invitation).

Scleroderma—Progressive Generalized Type? Presented by DR. S. BLAU and DR. N. KOS (by invitation).

Case for Diagnosis: Granuloma Annulare; Erythema Perstans? Presented by DR. A. J. ORFUSS and DR. JAMES ROCHFORD (by invitation).

PHILADELPHIA DERMATOLOGICAL SOCIETY

Herman Beerman, M.D., Chairman
Herbert A. Luscombe, M.D., Secretary

Jan. 16, 1959

Lymphocytoma Cutis (Follicular Lymphoma?). Presented (by invitation) by DR. FREDERICK URBACH and DR. J. GOLDSTEIN.

The patient, a white man aged 52, of good general appearance, presented on the left shoulder several reddish-purple nodules, firm and poorly demarcated. These formed an irregular circinate strip of nodular infiltrate. Previous biopsies showed lymphocytoma cutis. The condition began some 15 years ago as an isolated pink nodule on

the left arm, gradually progressing to form nodules and irregular half-circles. The patient has been treated by Dr. Tulipan, in New York, and Dr. Pillsbury, in Philadelphia, with roentgen ray, grenz ray, prednisone, and triamcinolone. He has had no treatment in the past two months, and there has been recurrence of lesions, associated with itching and burning.

The complete blood count was within normal limits.

The report on the biopsy specimen was "Lymphocytoma cutis (follicular lymphoma?)"

The patient has been treated with 9,000 r of grenz ray as well as prednisone and triamcinolone.

Discussion

DR. J. GRAHAM (by invitation): I never had a chance to study the section until this evening, but on the basis of what I see, I would certainly have to agree with the diagnosis of malignant lymphoma, and it seems to be of the reticulum cell type. There is a monomorphous type of cell; there is some variation inside, but it appears to be of the same type; there are a lot of mitotic figures; and I do not see how I could call it anything else than lymphocytoma cutis.

DR. WALTER B. SHELLEY: Dr. Pillsbury and I observed and treated this patient from 1951 through 1957. We have seen this group of lesions regress, under grenz as well as roentgen ray therapy, only to reappear. Low dosage systemic steroids have had a similar masking effect. At no time have we been able to do anything in a definitive curative way. Three representative biopsies during our six-year period of observation were reported as: lymphocytoma cutis (Spiegler Fendt type), 1951; lymphocytoma cutis, 1955; lymphoma, indeterminate type, 1956. We feel that the prognosis here must remain guarded.

DR. GEORGE W. HAMBRICK JR.: Are there actual lymph follicles in this lesion?

DR. J. GRAHAM (by invitation): No, I did not see the follicular type.

DR. GEORGE W. HAMBRICK JR.: In spite of the current trend by some to group a large number of conditions under the one term lymphocytoma cutis, I believe that it is still worth while to separate these conditions, both on clinical and histopathological differences. The semicircular arrangement of large erythematous nodules certainly suggests lymphosarcoma clinically. The absence of follicle formation certainly rules against this being lymphocytoma cutis.

DR. FREDERICK URBACH (by invitation): The question here is one of therapy. Every time there is suppression, with regression of the lesions. Should we recommend that this be widely excised and grafted, which is the only other thing we can do now? However, if this is one of the lymphomas, it is doubtful that this should be recommended.

DR. JOHN F. WILSON: I think one has to remember that one is not curing a disease such as this, and that the treatment is purely palliative. One must be certain that one does nothing harmful to the patient, and that one keeps him alive as comfortably as possible for as long as one can. I personally have made it a practice to wait and see what happens to these patients, and if they get into difficulty, then carry out the treatment.

DR. GEORGE W. HAMBRICK JR.: I disagree with the foregoing speaker. There are patients with localized lesions of mycosis fungoides, Hodgkin's disease, or lymphosarcoma, who have lived for many years following drastic surgery. I favor such an attempt in patients such as the one under discussion.

Squamous Cell Carcinoma, Formerly Presented as Keratoacanthoma. Presented by CAPTAIN JAMES H. LOCKWOOD (MC), USN, and (by invitation) LT. D. L. BAXTER, (MC), USN.

The patient, a white man aged 25, had no significant childhood or adult illnesses prior to the present one. There is no history of antecedent trauma. His father died at the age of 47 with squamous cell carcinoma of the nose.

In August, 1947, this patient developed a papular lesion on the tip of his nose; this gradually enlarged to develop into a crateriform lesion 1.5 cm. in diameter, with a keratotic central area. The lesion was initially diagnosed as a keratoacanthoma, supported by three biopsies. However, it was felt most expedient to excise the entire lesion, and in November, 1957, this was done by the Plastic Surgery Service of the Naval Hospital. Frozen section revealed the true nature of this lesion as a squamous cell carcinoma, and the entire nose and anterior septum were resected. This was followed by bilateral neck dissection, in January and February, 1958, in two stages, six weeks apart. No metastatic lymph nodes were found. Subsequently he has had plastic reconstruction of his nose in multiple stages, and is currently in process of final shaping of his reconstructed nose. No metastases have been demonstrable since the initial excision of the nasal lesion. The patient is presented to demonstrate the hazards inherent in the diagnosis of keratoacanthoma without complete removal of the lesion for microscopic examination.

Laboratory examinations revealed no significant abnormalities.

Reports on biopsy specimens were as follows:

"Sept. 26, 1957. Lackbourne Air Force Base S57-2924. Sections of the original paraffin block, cut to three levels, showed fragment of skin exhibiting squamous hyperplasia, acanthosis, and

hyperkeratosis. There were clefts filled with keratin, and cyst-like spaces containing keratin in the epidermis. Within the dermis there were focal areas of necrosis, numerous epidermal appendages, and an intense inflammatory infiltration with plasma cells, lymphocytes, and occasional polymorphonuclear cells. Diagnosis: compatible with keratoacanthoma."

"Nov. 8, 1957. United States Naval Hospital, Philadelphia, S57-2798. The specimen was covered by hyperplastic, keratotic, well-differentiated, stratified squamous epithelium, containing occasional cyst-like spaces filled with keratin, and showing deep downgrowths in some areas. Beneath these downgrowths were deeply scattered clusters of well-differentiated stratified squamous epithelium. Scattered throughout the dermis there was marked infiltration with lymphocytes and plasma cells. Diagnosis: Keratoacanthoma."

"Nov. 18, 1957. United States Naval Hospital, Philadelphia, S57-2870. Rebiopsy of the nasal lesion shows hyperkeratosis, acanthosis, and, beneath the epidermis, dense infiltration with chronic inflammatory cells, chiefly plasma cells. Diagnosis: compatible with keratoacanthoma."

"Nov. 25, 1957. United States Naval Hospital, Philadelphia, S57-2925-A-B-C-D. Excisional specimen. The sections submitted for frozen section showed a neoplastic invasion extending to and involving the nasal septal cartilage, consisting of nests, sheets, and finger-like projections of squamous cells. The cells appeared to have distinct intercellular bridges, and exhibited hyperchromatism, anisonucleosis, individual cell keratinization, and frequent mitotic division figures. A few keratin pearls were observed. There were focal areas of hemorrhage and necrosis, together with an associated scattered infiltration with polymorphonuclear cells, lymphoid cells, and eosinophils. Similar findings were noted in the fixed tissue sections. Diagnosis: squamous cell carcinoma, nose."

"Dec. 20, 1957. United States Naval Hospital, Philadelphia, S57-3159, A to H. Eight separate biopsies from strategic superficial areas about the resected nose. No evidence of residual malignancy was seen."

"Jan. 21, 1958. United States Naval Hospital, Philadelphia, S58-164F and S58-188 ABCD. Specimen from left radical neck dissection, including 33 cervical lymph nodes and left sternomastoid muscle, showing only reactive hyperplasia of the lymph nodes, with no evidence of malignancy."

"Feb. 25, 1958. United States Naval Hospital, Philadelphia, S58-535 AB. Specimen from right radical neck dissection, including 40 cervical lymph nodes and the right sternomastoid muscle, showing only reactive hyperplasia of the lymph nodes, with no evidence of malignancy."

"Sept. 12, 1958. United States Naval Hospital, Philadelphia, S58-2396F. Biopsy of mucous membrane and nasal septum. No evidence of residual malignancy is seen."

Discussion

DR. HERMAN BEERMAN: This patient had a rather radical excision, which showed very active prickle cell carcinoma. His father had a carcinoma in the same site. This case emphasizes what I have been trying to get across: that one cannot diagnose keratoacanthoma under any circumstances with any degree of confidence, except in such an instance as Dr. Pillsbury and I reported (Multiple Keratoacanthoma, *Am. J. Med. Sci.* 236:614-623 [Nov.] 1958), in which the patient developed lesions under our eyes which could not be anything but keratoacanthoma.

Dermatitis Herpetiformis? Pemphigus Vulgaris? Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. B. SINA, DR. FREDERICK URBACH and DR. J. GRAHAM.

Cicatrizing Lichen Planus. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. FREDERICK URBACH and DR. J. GRAHAM.

Mycosis Fungoides. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. J. GOLDSTEIN, DR. FREDERICK URBACH, DR. J. GRAHAM.

Angiomatosis? Helioglobulinemia? Presented (by invitation) by DR. FREDERICK URBACH and DR. J. GOLDSTEIN.

Porphyria Cutanea Tarda (Chronic Mixed Hepatic Type—Watson). Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. J. GOLDSTEIN.

Keratosis Follicularis (Darier's Disease). Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. G. DIRKSSON.

Sclero-Dermatomyositis. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. R. H. HIGH.

Histiocytic Granulomatous Insect Bite Reaction. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. FREDERICK URBACH, DR. J. GRAHAM, and DR. B. SINA.

1. Lichen Sclerosus et Atrophicus. 2. Parapsoriasis Varioliformis (Mucha-Haberman). Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. B. SINA, DR. FREDERICH URBACH, and DR. J. GRAHAM.

Necrobiosis Lipoidica Diabeticorum? Diabetes Mellitus. Presented by DR. ALFRED TROSOW, and (by invitation) DR. J. GOLDSTEIN.

Sarcoidosis. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. FREDERICK URBACH, DR. J. GRAHAM, and DR. D. REBELLO.

Fibro-Xanthoma? Rule Out Granular Cell Myoblastoma. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. B. SINA, DR. FREDERICK URBACH, and DR. J. GRAHAM.

Tuberculosis Verrucosa Cutis. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. B. SINA, DR. FREDERICK URBACH, and DR. J. GRAHAM.

Granulomatous Inflammation. Miescher's Granulomatosis? Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. B. SINA, DR. FREDERICK URBACH, and DR. J. GRAHAM.

A Case for Diagnosis. Presented by DR. CARROLL F. BURGOON, and (by invitation) DR. FREDERICK URBACH, DR. J. GRAHAM, and DR. J. GOLDSTEIN.

SAN FRANCISCO DERMATOLOGICAL SOCIETY

R. Raymond Allington, M.D., President
Ralph T. Behling, M.D., Secretary
Herbert L. Joseph, M.D., Editor

Feb. 20, 1959

Subacute Disseminated Lupus Erythematosus with Endarteritis. Presented by DR. JOHN H. EPSTEIN and DR. FRANCIS J. SULLIVAN (by invitation).

A 14-year-old Nicaraguan school girl developed a facial eruption, purpura, and pain in the right hip in May, 1957. She was treated with atabrine for three months by her local physician and underwent a six-month remission. Severe repeated epistaxis, and normocytic, normochromic anemia developed in November, 1957. She was admitted to San Francisco General Hospital, where two L.E. preparations were positive, and the platelet count was 58,000. Prednisone, 100 mg. and chloroquin, 250 mg. daily. Transfusions were given. Steroids were gradually discontinued, and she did well until April, 1958, when purpura and thrombocytopenia reappeared, and prednisone therapy was reinstituted. She again improved, and steroids were withdrawn over a two-month period. She has since been maintained on chloroquin, 250 mg. daily.

Three months ago, discrete, erythematous nodules developed on the tips of the fingers and toes, associated with Raynaud's phenomenon. She has also exhibited finely scaled erythematous lesions of the periorbital and paranasal areas in the past two months.

The very extensive laboratory studies, in summary, showed the following positive results: sedimentation rate 29 mm/hr.; serum electrophoretic pattern: albumin 54.9%, alpha-1 3.2%, alpha-2 8.3%, beta 9.0%, gamma 24.5%; A/G ratio 4.1/3.8; F-2 test positive 1:448; 1+ albuminuria. L.E. preparations were negative in October and February.

Discussion

DR. FRANCIS J. SULLIVAN (by invitation): The L.E. test became negative in this patient after many months of medication with chloroquin. Apparently, the L.E. test is not a good indicator of the clinical course of the disease. Possibly, the high concentration of chloroquin in the patient's white cells affects the results. It would be interesting to use this patient's serum in an L.E. test with other white cells.

DR. JOHN H. EPSTEIN: This girl presents several interesting and unusual features. Endarteritis, as seen here, in lupus erythematosus, also occurs in other collagen diseases, notably rheumatoid arthritis and scleroderma. At one time, the question was entertained whether this might represent chilblains, or lupus pernio. It began in winter, and represents peripheral vascular dysfunction and obliteration. Raynaud's phenomenon is seen in about 10%-15% of patients with systemic lupus. In this instance, it was associated with the onset of the endarteritis last December, and has persisted, although the weather in San Francisco has not been cold by Eastern standards. It will be interesting to see what happens during the warmer months.

Rather typical dysproteinemic changes are seen. The positive F-2 test is an indication of dysproteinemia in the gamma globulin fraction which mainly has been found in rheumatoid arthritis, but approximately 16% of patients with systemic lupus show this change. The degree of positivity of the F-2 test goes much higher in rheumatoid arthritis than in lupus.

Case for Diagnosis: Bullous Eruption in a Child. Presented by DR. WALLACE PROCTOR.

A two-year-old boy has had a vesicular and bullous eruption of the head, face, and genitalia for six months. He is an adopted child, and was well until August, 1958, when clear vesicles first appeared on the face, and subsequently spread to involve the ears, scalp, neck, and scrotum. Itching is not a prominent symptom until the eruption becomes excessive, or infection develops.

When first seen, in November, 1958, there were erythematous crusted papular lesions on the cheeks, dry scaly patches, and a few pustules on the ears. The entire scrotum was red, and covered with superficial vesicles and pustules 3-4 mm. in diameter. On the lower abdomen were a few dry erythematous papules and blotches. Similar lesions were present on the upper thighs. The clinical impression at that time was pyoderma, or possible bullous impetigo.

Antibiotic sensitivity tests were done, and the indicated topical and oral antibiotics were prescribed against the coagulase positive Staphylococcus aureus cultured from a pustule. No growth was obtained from the clear vesicles. All other laboratory studies are essentially negative. There is 5% eosinophilia in the differential count.

The vesicles occur in recurrent crops, and some become infected. They regress in a few days, and

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are followed by new ones in adjacent areas. The general health has remained quite well. He was hospitalized for one week in December, 1958, but intensive treatment with compresses and antibiotics produced no improvement. He previously had been treated unsuccessfully with topical hydrocortone and an elimination diet.

A therapeutic test with sulfapyridine, 3¼ gr. (.25 gm.) twice daily, was continued for six days without benefit. Gradually the condition has become less severe.

Discussion

DR. NORMAN E. LEVAN (by invitation): This young patient brought to mind several similar instances in which the same diagnostic and therapeutic problems were considered.

At the onset, the eruption may suggest bullous erythema multiforme, but the prolonged course, and absence of a clear-cut response to steroids, point away from this diagnosis. I favor dermatitis herpetiformis, or a variant thereof, and refer the presenters to several excellent clinical reports on this subject of bullous dermatoses of infants in the *Brit. J. Dermat.*—particularly "Dermatitis Herpetiformis in Children" by Dr. Chaim Berlin (*Brit. J. Derm.* 71:281), and another by Dr. G. B. Dowling. Dr. Dowling described a group of cases which he would place in a separate category chiefly because of (*a*) their appearance soon after birth, (*b*) tendency for some of the lesions to be elicited by trauma, and (*c*) failure to respond well to sulfapyridine or diaminodiphenylsulfone.

The clinical picture is generally dominated by vesicles and bullae, widely disseminated, often arising from apparently normal or only slightly inflamed skin, and tending to form arcuate or circular plaques. These blebs are subepidermal—as in the present case. The prognosis is for recurrences to become gradually milder and less frequent. Hence, the use of such potentially toxic agents as sulfonamides or corticosteroids is perhaps best reserved for unusually severe exacerbations.

DR. LOUIS H. WINER (by invitation): As Dr. Levan indicated, this is a subepidermal bulla, the floor of which is formed by the cutis, and there are numerous eosinophils throughout the floor. It is a tension blister, histologically and clinically.

My impression is the same as Dr. Levan's—that there are two types of dermatitis herpetiformis—one in the adult, and one in the child. I think the prognosis in children is better than in adults. They seem to outgrow the disease, and tolerate sulfapyridine for years without ill effect.

DR. HERBERT L. JOSEPH: I suggested that Dr. Proctor try sulfapyridine, and this failed. Diasone might be tried, but the proper dosage must be determined for such a small child.

It is my impression that dermatitis herpetiformis eventually disappears spontaneously, but it takes many years. I can recall at least three patients who have had the disease for more than ten years. One of these, a young mother, now aged 25, improved on sulfapyridine when treated at the University of California ten years ago. Subsequently she has cleared completely during three pregnancies. Now she develops lesions only premenstrually, confined to the face as tiny, intensely pruritic vesicles. She is well controlled with one diasone tablet twice daily. In my experience, diasone is safer and more effective than sulfapyridine.

Case for Diagnosis: Lichen Sclerosis et Atrophicus; Atrophoderma of Pasini and Pierne; Morphea? Presented by DR. JAMES P. MCGINLEY and DR. EDWARD M. MOSS (by invitation).

Dermatitis Nodularis Necrotica. Presented by DR. MAX R. GREENLEE (by invitation).

Case for Diagnosis: Disseminated Lupus Erythematosus? Presented by COL. RAYMOND F. WILLIAMS, MC., U.S. ARMY, and CAPT. ALBRECHT F. J. SOMMER, MC., U.S. ARMY (by invitation).

Mycosis Fungoides Treated with Electron Beam. Presented by DR. HAROLD M. SCHNEIDMAN and DR. DONALD H. PAULSON (by invitation).

Abstracts from Current Literature

STUDIES OF SULPHUR, PHOSPHORUS AND SOME METALS IN SKIN FOLLICLES. M. L. RYDER, J. Histochem. 7:133-138 (March) 1959.

Radioactive sulfur, phosphorus, copper, zinc, and potassium were administered to experimental animals. Autoradiographs were used to demonstrate the localization of the radioactivity in the hair. Sulfur enters the follicles rapidly, and its appearance in the keratinized portion of the hair in 24 hours in the rat and mouse and 48 hours in the lamb suggests diffusion of sulfur-containing compounds.

Copper was not consistently incorporated into any portion of the hair follicle. Zinc was concentrated in the unkeratinized part of the follicle, especially just above the bulb. Potassium localized in the stratum corneum, follicular orifice, and the sweat duct. Also potassium was found in the outer root sheath in the lower part of the follicle. Phosphorus was mainly present in the bulb.

SMITH JR., Miami, Fla.

NEVUS COMEDONICUS. H. BEERMAN and J. B. HOMAN, Arch. klin. u. exper. Dermat. 208:352-342, 1959.

This article is a review of the literature and a report of eight new cases of the authors' own observation. Nevus comedonicus occurs with the same frequency in both sexes. In females no lesions were observed below the costal margin on forearms and hands; in males the right side of the body below the costal margin was affected more frequently, whereas no lesions were found on the forearms and hands. Comedos in nevus comedonicus differ from comedos in acne vulgaris, since the former show active changes of the hair follicles in form of acanthosis and hyperkeratosis. There is no satisfactory therapy except for deep surgical removal of small lesions.

HOLLANDER, Springfield, Mass.

PRESENCE OF LACTIC DEHYDROGENASE IN PATHOLOGIC HUMAN STRATUM CORNEUM. G. WEBER, Arch. klin. u. exper. Dermat. 208:362-366, 1959.

Lactic dehydrogenase was found in high concentration in pathologic material of stratum corneum, particularly in psoriasis vulgaris and dry eczema. There also was increased activity of the enzyme, but to less degree, in scales of normal skin after repeated ultraviolet irradiation.

HOLLANDER, Springfield, Mass.

DIAGNOSIS AND THERAPY OF MYCOSES DUE TO IMPERFECT YEASTS (CRYTOCOCCACEAE). H. RIETH and J. SCHÖNFELD, Arch. klin. u. exper. Dermat. 208:343-361, 1959.

Authors computed six tables for identification of the various species of crytococcacae, such as Candida, Torulopsis, Cryptococcus, Rhodotorula, and Trichosporon. In situ microscopic examination of rice agar cultures in Petri dishes and serological identification by the method of Tsuchiya are helpful diagnostic means. Amphotericin B is recommended in the therapy of cryptococcosis and nystatin in the therapy of mycoses caused by Candida, Torulopsis, and Trichosporon.

HOLLANDER, Springfield, Mass.

RETICULOSIS AS A TYPE OF ABT-LETTERER-SIEWE DISEASE. C. EBERHARTINGER and R. SANTLER, Arch. klin. u. exper. Dermat. 208:367-379, 1959.

The authors describe a case of reticulosis in a 14-month-old boy. There were intermittent fever, xanthoma-like lesions of the face, papulosquamous, partly necrotic, and hemorrhagic skin changes of the scalp and trunk, and diffuse erythematous, oozing skin of intertriginous regions. Enlargement of various lymph nodes and osseous defects of the occipital part of the cranium were also found. Histologic examination of the skin showed in the upper and middle cutis band-like infiltrates of reticular elements, lymphocytes, eosinophils, and a few giant cells. Lymph nodes also revealed reticular elements. Topical treatment with antibiotics and hydrocortisone was not successful. Achromycin by mouth

decreased the fever but had hardly any influence on skin changes. Oral cortisone was also without effect on the skin.

HOLLANDER, Springfield, Mass.

FREE PURINES AND FREE PHOSPHATE IN AQUEOUS EXTRACTS OF CALLUS AND PSORIASIS SCALES. O. BRAUN-FALCO and K. SALFELD, Arch. klin. u. exper. Dermat. 208:395-401, 1959.

Psoriasis scales in comparison with callus showed increased values of free purines and particularly of free phosphate. These changes seemed to be characteristic but not specific for psoriasis.

HOLLANDER, Springfield, Mass.

BEHAVIOR OF PENTOSES AND POLARGRAPHICALLY REDUCING SUBSTANCES OF KERATOTIC EPIDERMIS IN NORMAL AND PATHOLOGIC KERATOSIS (PSORIASIS VULGARIS). TH. GRÜNEBERG and A. SZAKALL, Arch. klin. u. exper. Dermat. 208:402-409, 1959.

Psoriasis scales and stratum corneum conjunctum material of normal skin were examined. Psoriasis scales showed higher value of pentoses than normal stratum corneum conjunctum material. Polarographically reducing substances were found in normal keratotic material but not in psoriasis. Paper chromatography showed a distinct difference between normal stratum corneum conjunctum material and psoriasis. The authors interpret their findings as quantitative and qualitative changes of epidermal cell metabolism in psoriasis.

HOLLANDER, Springfield, Mass.

IMPETIGO HERPETIFORMIS-PSORIASIS PUSTULOSA-ACRODERMATITIS CONTINUA HALLOPEAU. P. MÖSLEIN, Arch. klin. u. exper. Dermat. 208:410-458, 1959.

Four hundred cases from the literature and seven cases of the author's own observation are reviewed. The author concludes that impetigo herpetiformis is an independent disease. All cases of psoriasis with localized or generalized pustule formation should be considered psoriasis cum pustulatione. Acrodermatitis continua Hallopeau is an independent disease. Some cases described as pustular psoriasis, type Barber, and localized acrodermatitis continua represent cases of pustular bacterid.

HOLLANDER, Springfield, Mass.

DEMONSTRATION OF ENZYMATIC MECHANISMS IN HUMAN EPIDERMIS ON THE MODEL OF SUBEPIDERMAL BULLA. G. WEBER and H. THEISEN, Arch. klin. u. exper. Dermat. 208:459-469, 1959.

Epidermis and serum of fresh bullae of pemphigus with subepidermal bulla formation were examined in two cases. The following enzymes and metabolites from anaerobic glycolysis and the citric acid cycle were demonstrated: aldolase, pyruvic acid, lactic dehydrogenase, lactic acid, glutamic acid-pyruvic acid-transaminase, adipic acid-dehydrogenase, α-ketoglutaric acid, glutamic acid-oxalic acid-transaminase, and glutamic acid. It was proved that these enzymes and metabolites are not only found in human epidermis but are produced by its cell layers. Comparative quantitative examinations revealed that glycolysis, citric acid cycle, and protein metabolism in connection with these systems occur in human epidermis.

HOLLANDER, Springfield, Mass.

MECHANISM OF GROWTH STIMULATION OF FUNGI BY PENICILLIN. D. JANKE, Arch. klin. u. exper. Dermat. 208:470-474, 1959.

Heat destroys growth-stimulating and antibiotic effects of penicillin on fungi. The stimulating effect is decreased with the use of almost melting point-pure penicillin. This effect is due to unknown, probably impure by-products of commercial penicillin G sodium. Changes of pH values of culture media can be excluded as cause of the stimulating effect. The chemical nature of the stimulating agent is not yet known; its pure demonstration and removal from commercial penicillin are to be desired.

HOLLANDER, Springfield, Mass.

SIGNIFICANCE OF LOCAL COLD ERYTHEMA FOR ECZEMA DIAGNOSIS. A. W. ENGELHARDT, Arch. klin. u. exper. Dermat. 208:475-488, 1959.

The macroscopically visible reaction of the blood vessels of the skin to cold (local application of ice cubes for 3 and 30 seconds, respectively) was observed in 436 patients: 33 with neurodermatitis, 10 with psoriasis vulgaris, 15 with eczematized psoriasis (inversa), 78

with seborrheic dermatitis, 20 with contact dermatitis, 16 without eczema, and 403 without neurodermatitis. There were increased cold erythema latency and shorter erythema duration in patients with neurodermatitis compared with the other groups of patients. In patients with psoriasis vulgaris, longer cold erythema latency was observed in contrast with shorter duration in patients with eczematized psoriasis.

HOLLANDER, Springfield, Mass.

CAPACITY OF SKIN SURFACE FOR SPLITTING OF ESTERS. G. K. STEIGLEDER and H. ELSCHNER, Arch. klin. u. exper. Dermat. 208:489-501, 1959.

With the use of a color test it was shown that the skin surface is capable to split esters. The nature of the ester-splitting substances of the skin surface cannot be determined; probably they are exoenzymes.

HOLLANDER, Springfield, Mass.

LABILE HYPERTONIA AND MANIFESTATION RHYTHM OF CHRONIC THROMBOCYTOPENIC PURPURA. H. HOLZMANN and G. W. KORTING, Arch. klin. u. exper. Dermat. 208:502-515, 1959.

Observation of intermittent chronic thrombocytopenic purpura in a 78-year-old man. There was a definite relation between low faces of labile hypertonia, decrease of blood platelets, and development of new purpuric lesions. This observation speaks in favor of a central nervous regulation of the blood platelet system.

HOLLANDER, Springfield, Mass.

CONTRIBUTION TO SYMPTOMATOLOGY OF ACRODERMATITIS CHRONICA ATROPHICANS (PICK-HERXHEIMER). C. DONNERMANN and H.-J. HEITE, Arch. klin. u. exper. Dermat. 208:516-527, 1959.

Eight hundred forty cases of acrodermatitis chronica atrophicans (ACA) are reviewed from the literature. ACA is observed two to three times more in women than in men. The beginning of the condition is seen most frequently in the fourth decade of life. Dermatosclerosis and ulceration are a frequent combination. Simultaneous appearance of fibroid nodules and ulceration is not reported. Fibroid nodules are more localized on the arms; dermatosclerosis occurs more on the legs; anetoderma does not show any favorite localization. Ulceration is usually seen on the legs and not on the arms. The author feels that ulcerations are not caused by ACA but are an accompanying symptom of dermatosclerosis.

HOLLANDER, Springfield, Mass.

NEW POSSIBILITIES OF SOFT TISSUE DIAGNOSIS WITH ELECTRONIC-COPY APPARATUS UNDER SPECIAL CONSIDERATION OF DERMATOSES. K. H. KÄRCHER and W. BADER, Arch. klin. u. exper. Dermat. 208:528-538, 1959.

The use of an electronic copy apparatus represents an improvement in the differentiation of soft tissues. Pictures of hyalinosis cutis, cavernous hemangioma, varicosis, and thrombophlebitis produced by this method are shown.

HOLLANDER, Springfield, Mass.

HISTOLOGIC AND HISTOCHEMICAL CHANGES IN TEMPORARY MONOIODOACETATE ALOPECIA. O. BRAUN-FALCO and H. THEISEN, Arch. klin. u. exper. Dermat. 208:539-558, 1959.

This is a discussion of histologic and histochemical examinations of skin and hair of 18 rats which received injections of monoiodoacetate solution. The cells of the hair matrix show a most sensitive reaction to monoiodoacetate. Less sensitive are the mesodermal cells of the papilla and the cells of the external root sheath. New hair growth again starts from the two latter cells. Disturbances of cellular metabolism, so-called dysfermentations of the active hair matrix, cause temporary alopecia.

HOLLANDER, Springfield, Mass.

THE PROBLEM OF INTERRELATION BETWEEN DIFFUSE GENERALIZED SCLERODERMA, ACROSCLEROSIS, RAYNAUD'S PHENOMENON AND RAYNAUD'S DISEASE. Z. STAVA, Dermatologica 118:1, 1958.

Study of 70 cases of diffuse scleroderma shows (1) that there is a very close relation between Raynaud's disease and scleroderma of the acrosclerotic type; (2) that Raynaud's disease precedes it; (3) that Raynaud's disease has no connection with acute malignant diffuse generalized scleroderma, and (4) that prognosis of acrosclerosis is less favorable the

earlier in life that Raynaud's phenomenon occurs and the shorter the interval between the onset and the appearance of sclerodermatous changes.

MONASH, Miami, Fla.

ANATOMY OF THE SKIN. H. PINCUS, Dermatologica 118:44, 1958.

Pincus gives his usual thorough review of the work on all phases of the skin published in 1956 and 1957.

MONASH, Miami, Fla.

EFFECT OF HYDROCORTISONE ON MITOCHONDRIAL MEMBRANE PERMEABILITY. C. H. GALLAGHER, Nature, London 182:1315-1316 (Nov. 8) 1958.

Hydrocortisone has been shown to inhibit severely the oxidative metabolism of mitochondria in vitro. This inhibition is due to the action of the steroid in increasing the permeability of the mitochondrial membranes. Loss of selective permeability allows the soluble cofactors of respiration to be leached from the mitochondria, thus inactivating pyridine nucleotide-linked dehydrogenases.

SMITH JR., Miami, Fla.

MELANIN: A NATURALLY OCCURRING CATION EXCHANGE MATERIAL. L. P. WHITE, Nature, London 182:1427-1428 (Nov. 22) 1958.

Because the physical characteristics of melanin of complex polymeric structure, insoluble particulate state, and ability to take up large amounts of water are also the chief characteristics of some of the synthetic organic ion-exchange resins, ion-exchange activity of partially purified melanin was examined. Melanin from both mammalian and invertebrate sources was demonstrated to have considerable activity as cation exchange material. Some cation exchange polymers can absorb electrons. Thus, the increase in melanin in the skin after exposure to sunlight or ionizing radiation suggests that the ion exchange activity of melanin may be one of its most important functions.

SMITH JR., Miami, Fla.

ACTIVITY OF 10-HYDROXYDECENOIC ACID FROM ROYAL JELLY AGAINST EXPERIMENTAL LEUKEMIA AND ASCITIC TUMORS. G. F. TOWNSEND, Nature, London 183:1270-1271 (May 2) 1959.

Mixture of royal jelly with tumor cells before inoculation completely suppressed the development of a transplantable mouse leukemia and the formation of ascitic tumors in mice. Fractionation studies established that this antitumor activity resided in the main fatty acid of the royal jelly, 10-hydroxydecenoic acid. Approximately 100 mg. of fresh whole royal jelly or 1.0 mg. of 10-hydroxydecenoic acid per ml. of cell-suspension was required for complete protection. At lower dosages, development of the tumors was delayed for five to six days, after which rapid tumor formation and death of all mice occurred. Acid pH and mixture of the cells prior to injection were necessary to demonstrate the activity. The dependence of the biological activity on an acid pH suggests that only the undissociated acid is effective.

SMITH JR., Miami, Fla.

HAIR AS A SUBSTRATE FOR NONKERATINOLYTIC FUNGI. D. M. GRIFFIN, Nature, London 183: 1281 (May 2) 1959.

Hair which has been autoclaved or sterilized with propylene glycol can support limited growth of many fungi which are not keratinolytic. Among other genera of the Fungi Imperfecti, Aspergillus, Penicillium, Alternaria, Curvularia, and Helminthosporium grow sparsely on the hair surface and produce spores there. Sterile human hair is placed on the surface of a thin layer of agar of low nutrient status contained in a Petri dish, and the agar inoculated with the fungus to be studied. Within a few days, the mycelium has spread from the agar to colonize sparsely the hair surface and scattered conidiophores are produced. The hair is carefully removed from the agar and mounted in any suitable mounting fluid. The advantage of the method is in the ease with which an undisturbed preparation of conidiophores and conidia may be prepared.

SMITH JR., Miami, Fla.

PENICILLIN TREATMENT OF GENERAL PARESIS (DEMENTIA PARALYTICA). R. D. HAHN, B. WEBSTER, G. WEICKHARDT, E. THOMAS, W. TIMBERLAKE, H. SOLOMON, J. H. STOKES, J. E. MOORE, A. HEYMAN, G. GAMMON, G. A. GLEESON, A. C. CURTIS, and J. C. CUTLER, A. M. A. Arch. Neurol. & Psychiat. 81:557-590 (May) 1959.

Repository penicillin is as suitable as aqueous penicillin for the treatment and prevention of general paresis. The general problem of dementia paralytica and its treatment is reviewed thoroughly.

LAZAR, Chicago.

POISON IVY DERMATITIS. R. J. LANGS, A. M. FUCHS, and M. B. STRAUSS, Indust. Med. 28:257-261 (June) 1959.

Eight guinea pigs were fed 600 times the human dose of Aqua Ivy tablets for 16 days. The animals were then found to be negative to patch tests, and postmortem examinations revealed no abnormalities. The poison ivy tablets in humans were followed by enhanced healing, and there were no side-effects or exacerbations. Sixty-eight persons with a history of poison ivy dermatitis received prophylactic therapy and challenging exposure ranging from almost daily exposures to once monthly. The incidence of poison ivy in this group was "considerably less than would be expected" in a similar unprotected group.

LAZAR, Chicago.

PRESERVATIVE AND PROTECTIVE CARE OF THE SKIN IN INDUSTRIAL WORKSHOPS. W. SCHNEIDER, M. HUBER, and W. POPP, Berufsdermatosen 7:1-11 (Feb.) 1959.

The prophylactic effect of a glycerin emulsion is compared with that of various "protective" creams. For the majority of the workers the glycerin emulsion proved to be adequate and usually preferable. Only in certain occupations where cutting oils, brake fluid, lacquers, and their solvents are handled, is additional use of protective preparations, such as silicone ointments, necessary.

BULEY, Champaign, Ill.

OCCUPATIONAL DERMATOSES DUE TO PHENOTHIAZINE DERIVATIVES. H. IPPEN, Berufsdermatosen 7:12-21 (Feb.) 1959.

Among the dermatoses caused by occupational contact with phenothiazine derivatives, the eczematous reaction is the commonest. In establishing the etiological relationship between the drugs and the skin eruption, the customary patch tests may not be sufficient, since the photosensitizing property of the drugs may be the important pathogenic factor. In such cases, the exposure of the patch-tested areas to suberythema doses of ultraviolet light will be of great diagnostic value.

BULEY, Champaign, Ill.

THE TREATMENT OF SUPERFICIAL MYCOSIS OF THE SKIN WITH GRISEOFULVIN. V. PARDO-CASTELLO, F. TRESPALACIOS, P. FARIÑAS, and G. F. BAQUERO, Bol. Soc. cubana dermat. sif. 16:1 (March) 1959.

From September, 1956, to October, 1957, the authors performed mycologic studies, adding 0.5 cc. of 1% solution of griseofulvin to 20 cc. of Sabouraud's medium and found that this antibiotic inhibited the growth of several dermatophytes. Since January, 1957, griseofulvin was used topically in the form of powder, cream, or solution in patients with superficial fungus infections. Because of the fungustatic properties of the drug, some improvement was noted, but in no case was a complete cure observed. In November, 1958, systemic medication was started. The doses used were 500 mg. to children less than 7 years of age, 750 mg. up to 14 years, 1,000 mg. to adults of average weight, and even up to 2,000 mg. to those over 150 lb. Thirty-seven patients were treated. The medication was ineffective in two cases of chromoblastomycosis and one of cryptococcosis. There were 13 cases of eczema marginatum, 2 of pityriasis versicolor, 6 of tinea capitis, 4 of onychomycosis, and 9 of dermatophytosis of the feet. The drug was effective in curing all superficial lesions in a period of two to three weeks, although the medication was continued for two weeks longer. In cases affecting the nails, from two to four months may be necessary before treatment is completely successful. In two patients symptoms of intolerance were observed, such as headache, abdominal cramps, and nausea, after the first few doses of griseofulvin. After the drug was discontinued, no further symptoms were observed and no changes in the blood could be detected. No renal

complications have been observed. The authors conclude that, at present, griseofulvin is the treatment of choice of all superficial mycosis.

CANIZARES, New York.

TREATMENT OF ZOOPARASITOSIS OF THE SKIN WITH PHENYLBUTAZONE. M. T. GONZALEZ and R. REVILLA Y RAMIREZ, Bol. Soc. cubana dermat. sif. 16:12 (March) 1959.

The authors had occasion to observe a patient with arthritis, pediculosis capitis, and scabies, who was cured of the two latter conditions without any local treatment following three days of administration of phenylbutazone (Butazolidin) prescribed for the arthritis. Animal studies in a veterinarian center showed that cattle and dogs were entirely cured of infestation with fleas and ticks in three days of treatment, the medications being administered at 15 mg. per kilogram. Human studies were performed at an institution where 42 patients from 13 to 20 years of age were treated. The dose used was from 400 to 600 mg. daily. No local treatment was used. Eighty per cent cures were observed in three to six days. This percentage could have been higher if some patients could have been treated more intensively. Only 2.5% of intolerance was noted. This low incidence of side-effects is attributed to the fact that the persons treated were young and, besides their skin infestation, healthy.

CANIZARES, New York.

PIEDRA: FIRST CASE OBSERVED IN CUBA. G. F. BAQUERO and F. TRESPALACIOS, Bol. Soc. cubana dermat. sif. 16:21 (March) 1959.

The authors report the first case observed in Cuba of piedra affecting the hair of the scalp of a white male. The authors believe that the causative organism of this case of white piedra is a new species which they denominated Trichosporon pardi. There are several photographs and photomicrographs of the culture.

CANIZARES, New York.

DISTURBANCES OF PIGMENTATION WITH CHLOROQUINE. J. L. C. DALL and J. A. KEANE, Brit. Med. J. 1:1387-1389 (May 30) 1959.

Three patients treated with chloroquine diphosphate noted bleaching of the hair, usually preceded by visual disturbances. Two of these also experienced increased sensitivity to strong sunlight. Freckles which normally appeared other summers failed to develop. It is suggested that chloroquine in toxic doses may interfere with the deposition of pigment or replace normal pigment.

LAZAR, Chicago.

LIPOID PROTEINOSIS URBACH-WIETHE (HYALINOSIS CUTIS ET MUCOSAE). C. EBERHARTINGER and G. NEIBAUER, Hautarzt 10:54-65 (Feb.) 1959.

A report concerning two siblings who suffered from lipoid proteinosis with extensive involvement of the skin and oral mucosa is presented. The endocranial calcifications, roentgenologically demonstrated in both cases, are considered a typical manifestation of the disease.

BULEY, Champaign, Ill.

EXPERIMENTALLY INDUCED ROENTGEN-RAY ACANTHOSIS IN ANIMALS. H. J. HEITE and H. HIRTE, Hautarzt 10:72-76 (Feb.) 1959.

The acanthosis test, previously used in experimental dermatology for other purposes, was employed as an indicator for the epidermal response of guinea pigs to roentgen rays. The extent and rate of development and regression of the acanthosis after irradiation depended on the dose, the wave length, and the degree of fractionation of the x-rays.

BULEY, Champaign, Ill.

ACUTE DERMATITIS DUE TO A POISONOUS MOTH. K. HIGUCHI and H. URABE, Hautarzt 10:79-82 (Feb.) 1959.

The authors report eight cases of a papular dermatitis caused by contact with Euproctis flava, a night moth found in various countries of the Far East. The antigen responsible for the eruption was found in dart-like spikes located on the tail of the moths and deposited with its eggs. Systemic allergic reactions may accompany the dermatosis which usually subsides after a few weeks.

BULEY, Champaign, Ill.

ABSTRACTS FROM CURRENT LITERATURE

RADIOACTIVE THREADS AND THEIR USE IN DERMATOLOGY. H. J. ENDRES and K. E. SCHEER, Hautarzt 10:84-87 (Feb.) 1959.

Radioactive phosphorus (P^{32}), a source of β-radiation, is placed as iron phosphate into hollow "Collafil" threads, an absorbable surgical suture material. These filaments, carrying 40μc.-100μc. per centimeter, are inserted with a needle into cavernous hemangiomas where they are slowly absorbed. In 78 cases treated in this manner the clinical results were satisfactory.

BULEY, Champaign, Ill.

SILICONE GRANULOMA OF THE SKIN. R. DEGOS and J. CIVATTE, Hautarzt 10:106-110 (March) 1959.

The coincidental occurrence of silicone granulomas and Besnier-Boeck-Schaumann sarcoid in two cases is reported. The clinical and histological similarity of the two dermatoses suggests a common etiology and pathogenesis.

BULEY, Champaign, Ill.

THE INFLUENCE OF THE CENTRAL NERVOUS SYSTEM ON THE COURSE OF ALLERGIC REACTIONS AND BACTERIAL INFECTIONS OF THE SKIN OF TEST ANIMALS. H. MIERZECKI, M. DEMIANOWSKA, A. WASIK, and A. WOYTON, Hautarzt 10:115-117 (March) 1959.

Guinea pigs were sensitized with dinitrochlorbenzole and aniline and, at the same time, exposed to electrical and mechanical shock. The results were equivocal: the allergic reactions were accelerated or delayed or followed the regular pattern. The treatment of the animals with neuroplegic agents resulted in delayed sensitization. Also, after experimental bacterial inoculation of the animals, the inflammatory response to infection was diminished, an observation which parallels clinical experience.

BULEY, Champaign, Ill.

CLINICAL OBSERVATIONS CONCERNING THE PATHOGENESIS OF CHLORACNE. W. BRAUN, Hautarzt 10:126-129 (March) 1959.

During a survey of an industrial plant manufacturing electrical condensors, 123 workers with chloracne due to chloronaphthalene were detected. In a number of cases, the distribution of the lesions seemed to indicate that direct contact with the compound and friction by clothing were responsible for the eruption.

BULEY, Champaign, Ill.

EXPERIMENTAL INFECTION OF MAN WITH TRICHOPHYTON VIOLACEUM. J. ESTEVES and H. NEVES, Hautarzt 10:151-154 (April) 1959.

Among 91 test persons inoculated with hair carrying Trichophyton violaceum, only 55 developed clinically manifest ringworm. After an incubation period of 4 to 24 days, the lesions produced the characteristic picture of tinea circinata but healed spontaneously after 11 to 79 days. In those persons who had suffered previously from a dermatomycosis, the eruption had a relatively shorter period of incubation, it spread less widely, and it involuted sooner. This behavior suggested an altered immunological state, although the testing with trichophytin failed to produce further evidence in this direction.

BULEY, Champaign, Ill.

THE DEVELOPMENT OF THE HAIR FOLLICLE IN MAN, IN PARTICULAR THAT OF THE INFUNDIBULUM AND THE CONNECTIVE TISSUE PORTION. H. PINKUS, Hautarzt 10:164-170 (April) 1959.

Contrary to the teachings of the past, the author proves that the epithelial portion of the hair follicle originates exclusively from the basal-cell layer of the embryo's epidermis, not by invagination of its entire epidermis. The layers of the hair follicle, therefore, are not homologous with the layers of the epidermis. The ectodermal and the mesodermal portions of the follicle organ grow and differentiate simultaneously and interdependently. The infundibulum develops froms the fetal hair canal which grows through the epidermis to its surface and is not the result of an epidermal differentiation. The significance of these new concepts in regard to dermatological pathology is discussed.

BULEY, Champaign, Ill.

THE SYNDROME OF ACUTE DISSEMINATED MONILIASIS IN ADULTS. A. I. BRAUDE, M.D., Ph.D., and J. A. ROCK, M.D., A.M.A. Arch. Int. Med. 104:91-100 (July) 1959.

Acute disseminated moniliasis in adults is characterized by the development of shock, fever, and gastrointestinal bleeding. This usually follows continuous antibiotic therapy during a protracted debilitating illness. It is postulated that susceptibility to Candida albicans occurs after alteration in bowel bacterial flora, gastrointestinal mucosal injury, and possibly hypocalcemia have occurred. Autopsy of a patient who died from acute disseminated moniliasis revealed multiple superficial intestinal ulcers filled with hyphae and spores. The endotoxin of C. albicans probably contributes to the development of shock.

LAZAR, Chicago.

OCULAR LESIONS IN XERODERMA PIGMENTOSUM. H. GILLER and W. C. KAUFMANN, A.M.A. Arch. Ophth. 63:130-133 (July) 1959.

Carcinoma of the cornea is presented in a case of xeroderma pigmentosum. Being of low-grade malignancy, the lesion of the cornea was treated by local excision. Few recurrences have been reported after such therapy.

LAZAR, Chicago.

SEVERE GASTROINTESTINAL HEMORRHAGE RESULTING FROM RECKLINGHAUSEN'S DISEASE. T. FARRAR and F. W. CHAPPELL, A.M.A. Arch. Surg. 79:106-109 (July) 1959.

Gastrointestinal tumors occur in patients with cutaneous neurofibromata. Persistent gastrointestinal symptoms may require roentgen examination or surgical exploration.

LAZAR, Chicago.

CAUSES OF CHRONIC URTICARIA INCLUDING INFECTION. A. CHESTER; H. LEIBOWITZ, and H. MARKOW, New York J. Med. 59:1786-1788 (May 1) 1959.

In two cases of chronic urticaria relief of symptoms continued for months after removal of a gangrenous gallbladder and appendix in one case and an inflamed appendix in the other. The role of infection in chronic urticaria is discussed.

LAZAR, Chicago.

HYPERSENSITIVITY TO COLD. N. S. BLACKMAN, New York J. Med. 59:2614-2617 (July 1) 1959.

A patient developed local and systemic hypersensitivity to cold one month after becoming hypersensitive to penicillin. Gradually increasing doses of histamine and exposure to water which is made cooler each time was of definite value in treatment. Fatal consequences to a systemic reaction or syncope may result, this being one cause of drowning.

LAZAR, Chicago.

HEMANGIOMAS. JEFF DAVIS, M.D., New York J. Med. 59:2574-2579 (July 1) 1959.

The author reviews the types of cutaneous hemangiomas, neurocutaneous syndromes associated with angiomatosis, and hemangiomas of other organs. He states that the complete adherence to a policy of postponing treatment of hemangiomas because they may undergo spontaneous regression is unwarranted.

According to the author, port wine marks may respond to thorium x, x-rays, or tattooing. Carbon dioxide is best for strawberry growths and resolution of the superficial portion of deeper tumors. In deeper tumors, sclerosing solutions give excellent results. Surgery, x-ray therapy, and radon seeds are indicated in selected situations.

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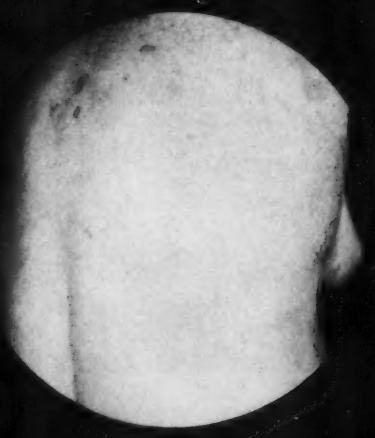
(1) Blank, H., and Roth, F. J.: A.M.A. Arch. Dermat. 79:259 (March) 1959. (2) Williams, D. I.; Marten, R. H., and Sarkany, I.: Lancet 2:1212 (Dec. 6) 1958. (3) Goldfarb, N., and Rosenthal, S. A.: Current M. Digest 26:67 (April) 1959. (4) Wrong, N. M.: Canad. M.A.J. *80*:656 (April 15) 1959.

Before treatment—tinea of the body of 9 years' duration. Infecting organism, **Trichophyton rubrum.**



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Photographs courtesy of Harvey Blank, M.D., Miami, Fla. The patients shown above received GRIFULVIN (griseofulvin, MCNEIL)

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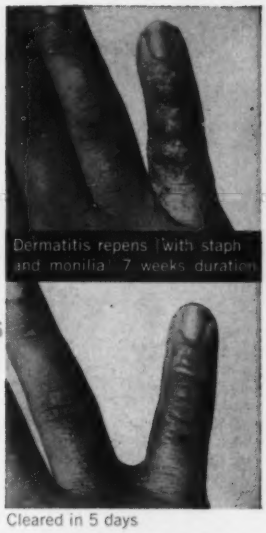

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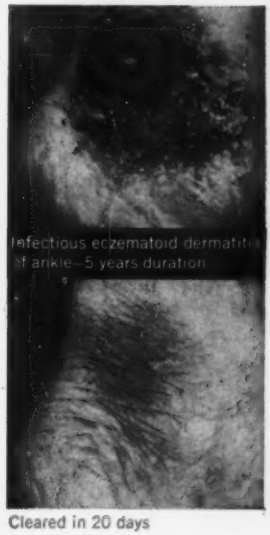

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References: 1. Shelmire, J.B., Jr.: Monographs on Therapy 3:164 (Nov.) 1958. • 2. Nix, T.E., Jr., and Derbes, V.J.: Monographs on Therapy 3:123 (Nov.) 1958. • 3. Robinson, R.C.V.: Bull. School of Med., U. Maryland 43:54 (July) 1958. • 4. Sternberg, T.H., Newcomer, V.D., and Reisner, R.M.: Monographs on Therapy 3:115 (Nov.) 1958. • 5. Clark, R.F., and Hallett, J.J.: Monographs on Therapy, 3:153 (Nov.) 1958. • 6. Smith J.G., Jr., Zawisza, R.J., and Blank, H.: Monographs on Therapy, 3:111 (Nov.) 1958. • 7. Monographs on Therapy, 3:137 (Nov.) 1958. • 8. Howell, C.M., Jr.: North Carolina M.J. 19:449 (Oct.) 1958. • 9. Bereston, E.S.: South. M.J. 50:547 (April) 1957.

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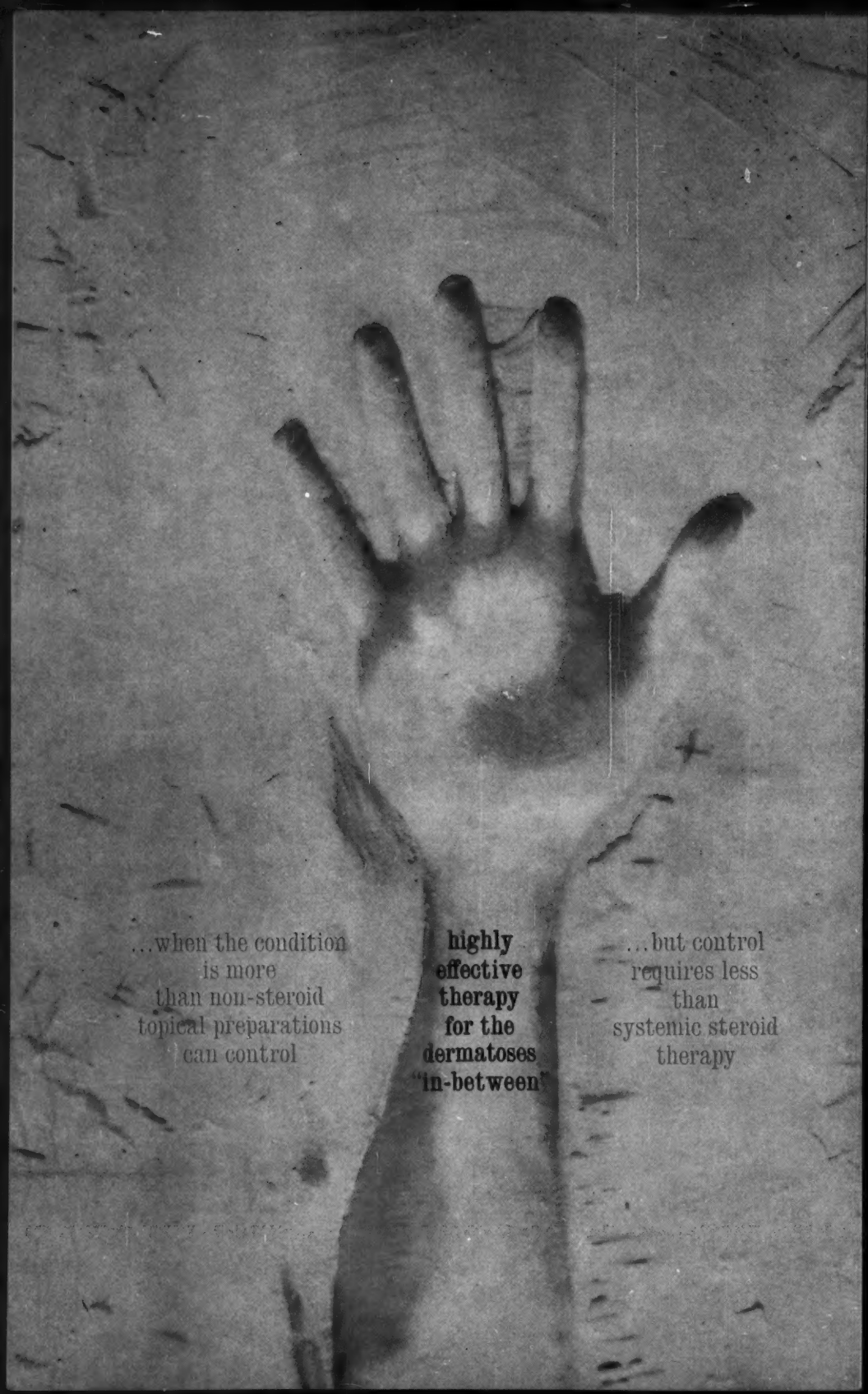
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*Baer, R. L., and Witten, V. H.: Editorial Comment, In The Year Book of Dermatology and Syphilology (1958-1959 Year Book Series). Edited by Rudolph L. Baer and Victor H. Witten, Chicago, The Year Book Publishers, 1959, p. 40.

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1. Shaw, J. M., and Crowe, F. W.: Skin Protective Ointments. Arch. Dermat. & Syph. 71: 379, 1955.

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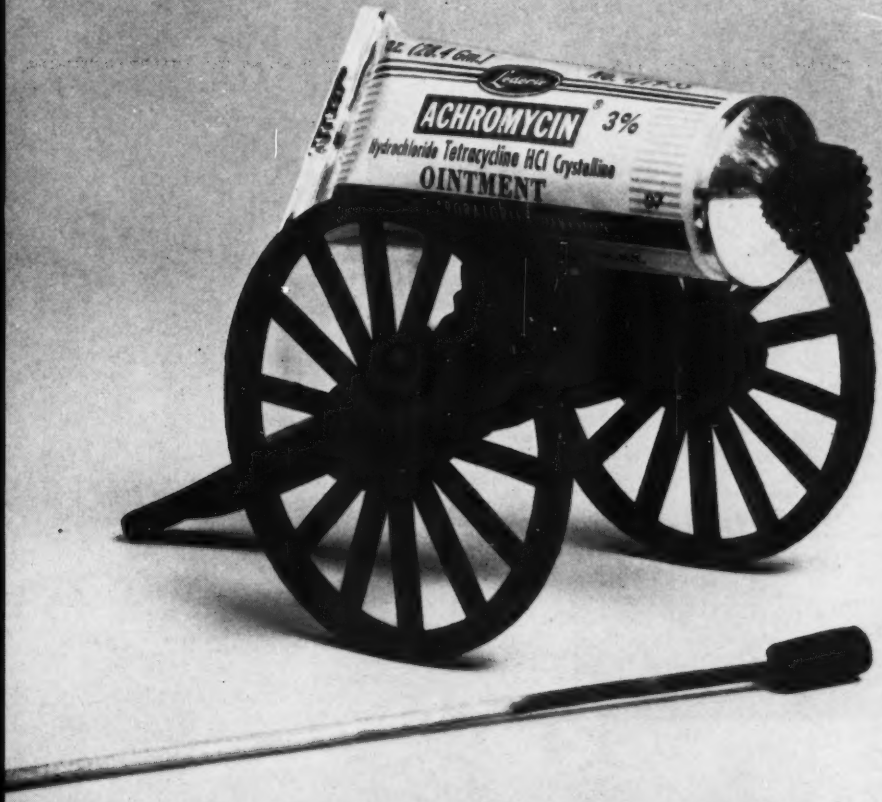

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*Diseases of the Colon & Rectum, Vol. 1, No. 5, Sept.-Oct. 1958.

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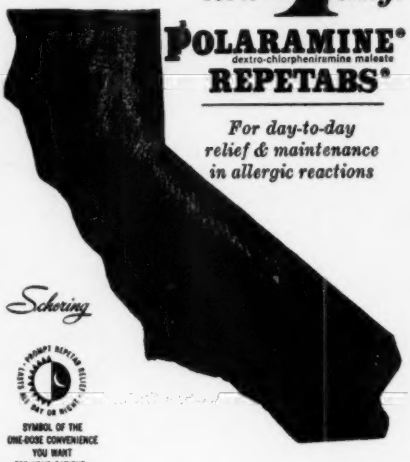

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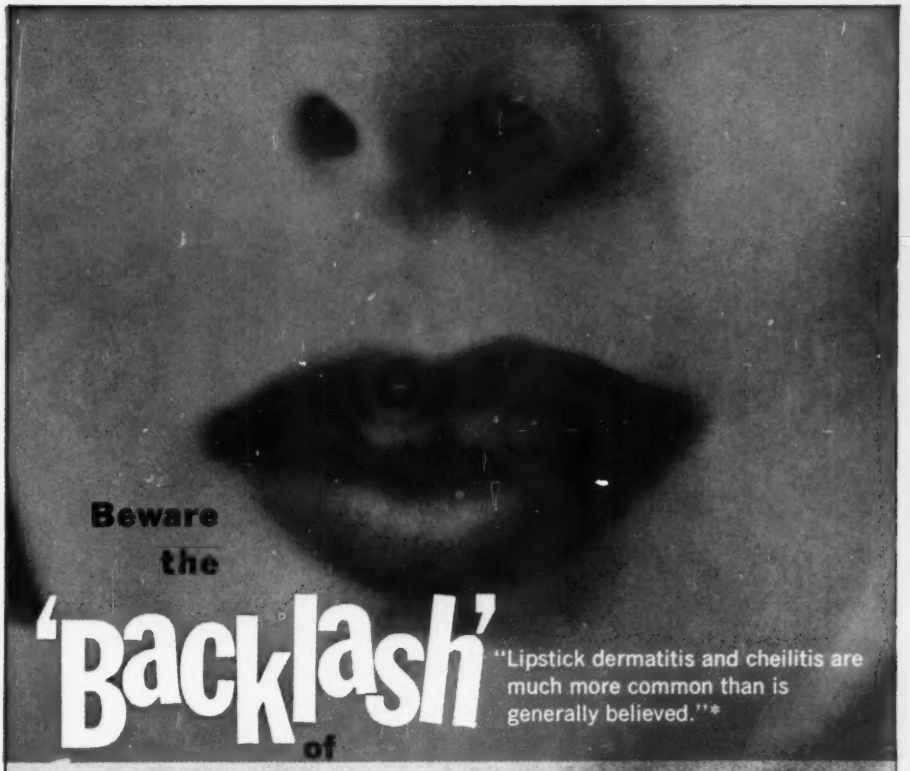

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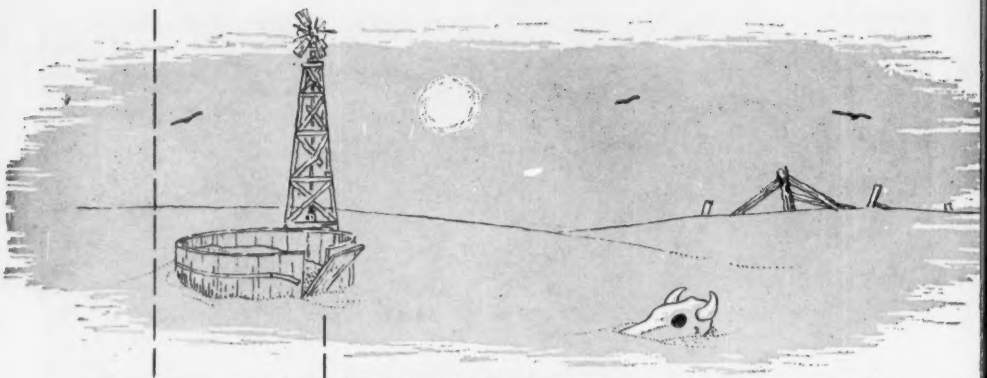

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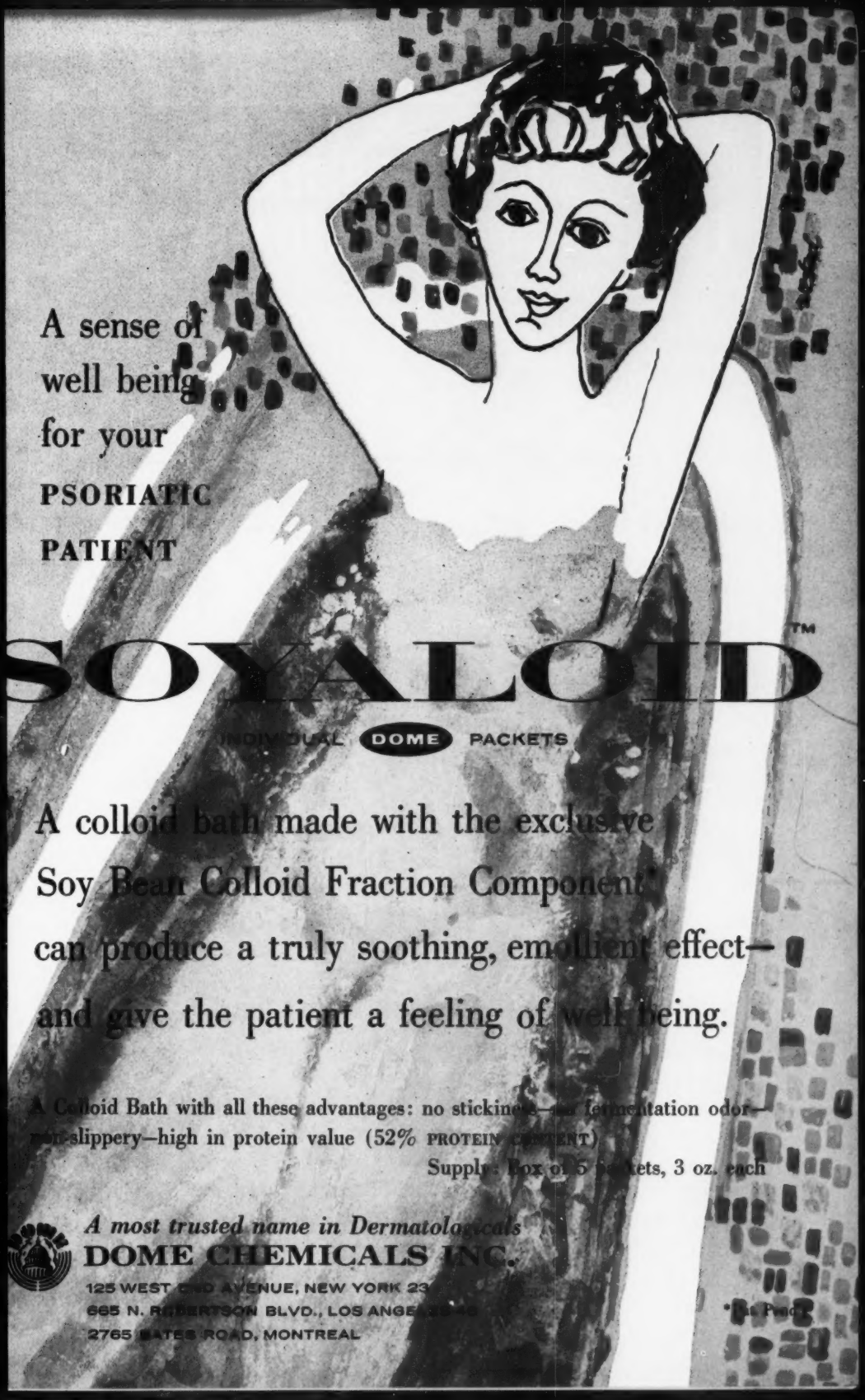
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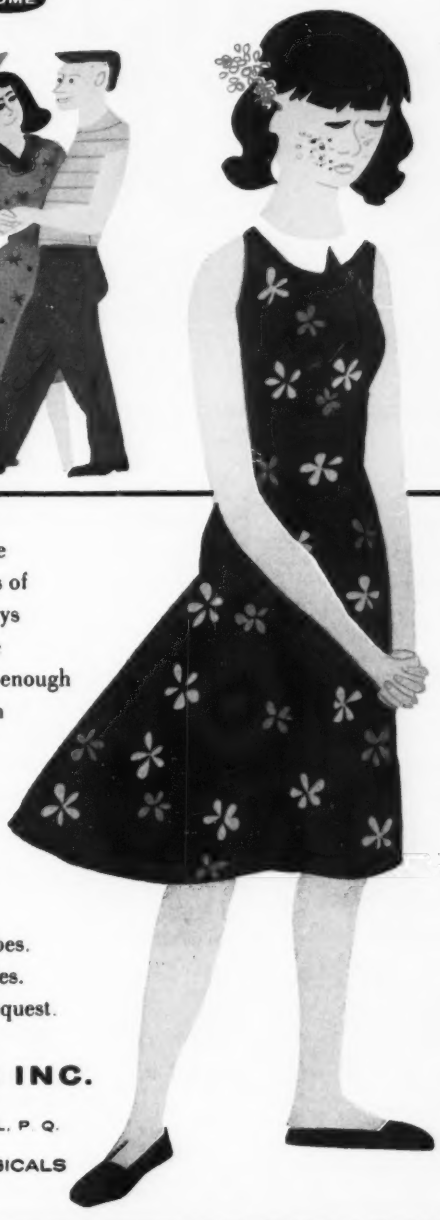

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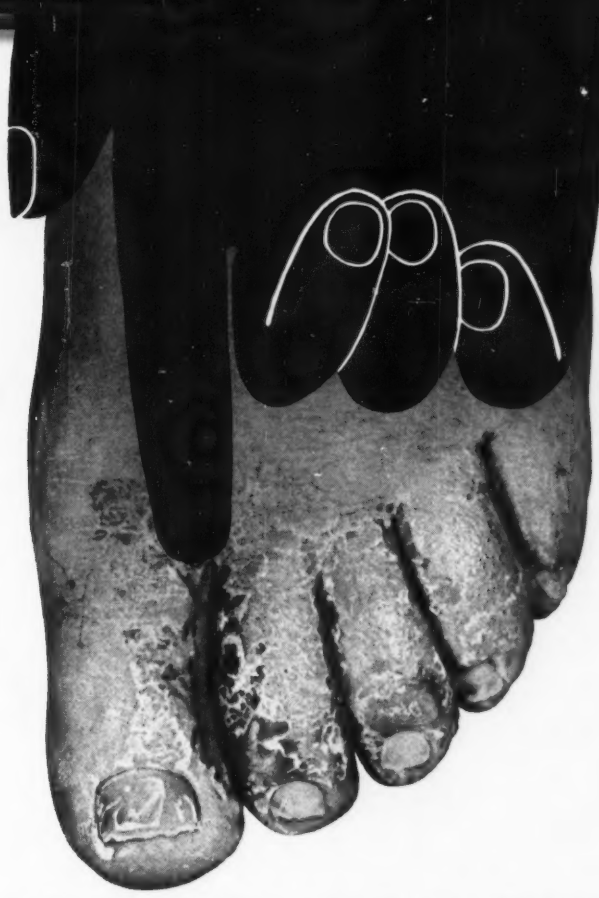

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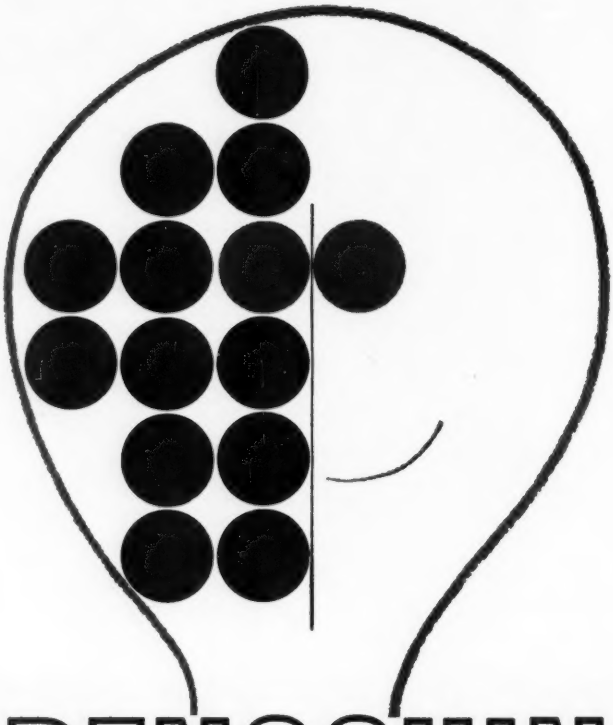

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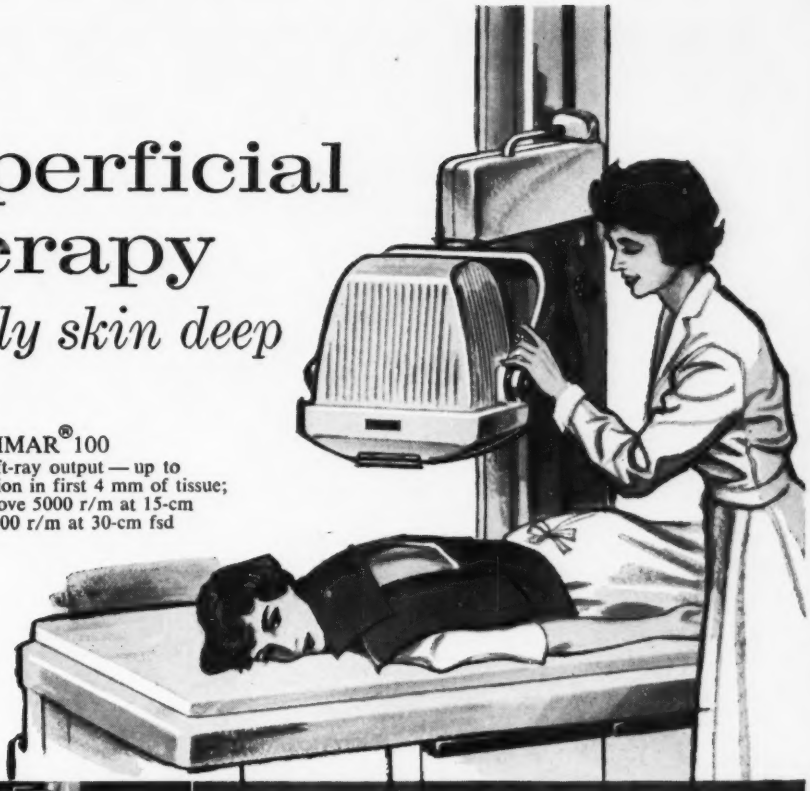

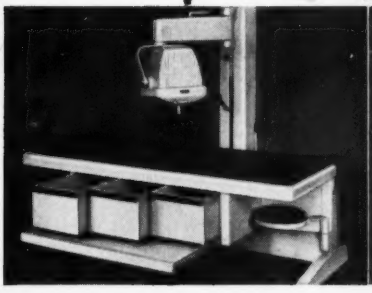

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Those times when *skin deep* is exactly *deep enough* — that's when you'll truly appreciate your Maximar 100. Foremost reason is its exclusive Coolidge tube with beryllium window: soft rays exit *in quantity* — are not filtered out as they would be with other materials. So Maximar 100's range easily extends from output approaching grenz rays to the depth penetration obtained at 100 kvp. Control is precise . . . dosage exact. And coverage at 30-cm focal/skin distance will span at least an 18-cm circle — perimeter output remaining at least at 80% of full center strength on an 18-cm circle! Talk over details of the Maximar 100 soon with your G-E x-ray representative. Or write X-Ray Department, General Electric Company, Milwaukee 1, Wisconsin, for illustrated Pub. P-112

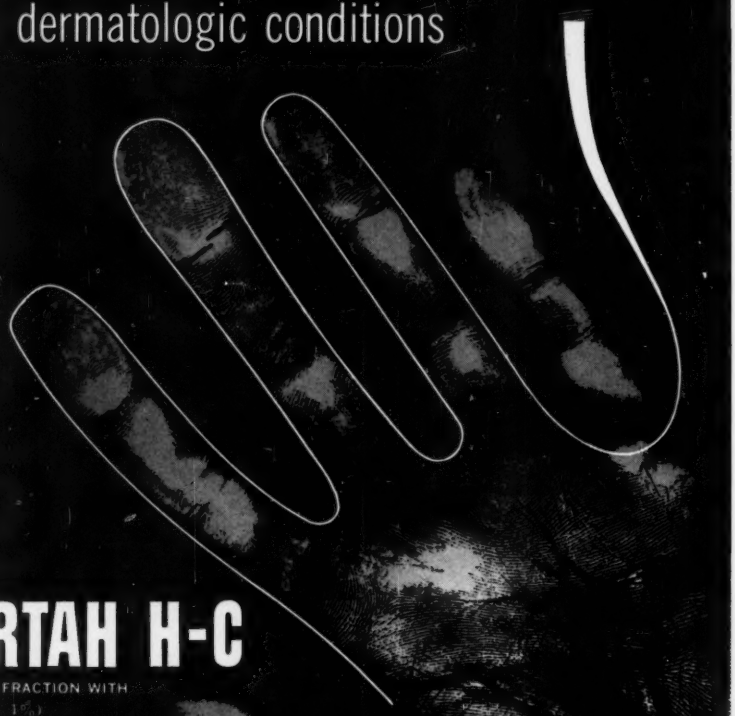
Progress Is Our Most Important Product

GENERAL ELECTRIC

spreads

THINLY · EVENLY · ECONOMICALLY

in acute dermatologic conditions



SUPERTAH H-C

(1.25% COAL TAR FRACTION WITH HYDROCORTISONE 1%)

antiinflammatory — antipruritic

- water miscible base . . . purified white tar fraction, stainless, washable
- maximum efficiency . . . spreads thinly for evenly distributed therapeutic action and great economy
- enhanced benefits . . . more effective than either agent alone
- excellent response . . . deep penetration to source of inflammation

supplied: ½-oz. tube

also available:

SUPERTAH Ointment (1½-oz. tube)
provides the therapeutic effectiveness of time-proved tar medication

SUPERTAH S/S (with salicylic acid and sulfur; 1½-oz. tube)
for keratolytic action

All Supertah preparations are made with refined natural coal tar fraction.

TAILBY-NASON COMPANY, INC. DOVER, DELAWARE

For the treatment of dry, scaly skin

LOWILA® EMOLLIENT

an effective skin moisturizer

moisturizes and softens the skin

promotes moisture retention

supplements natural skin oils

restores and maintains normal skin pH

Lowila Emollient hydrates the skin . . . keeps it soft and smooth

Lowila Emollient is a new, effective skin moisturizer for the treatment of dry, scaly skin. It contains an excellent keratin-moisturizing agent Kerohydric®, a crystal-clear, dewaxed oil-soluble fraction derived from lanolin.

Lowila Emollient moisturizes the skin and promotes retention of moisture. When applied to the skin, Lowila Emollient provides a partially-occlusive "breathing" film that: (1) supplies its own water to the dehydrated keratin layer, (2) prevents excessive evaporation when the atmosphere is dry, (3) allows the skin to absorb water from an atmosphere with normal humidity.

Lowila Emollient also supplements natural skin oils, restoring the protective action of skin lipids lost through effects of water, weather and detergents. In Lowila Emollient, Kerohydric is dispersed in a lactic acid buffered emulsion that restores and maintains normal skin acidity.

LOWILA EMOLLIENT

Indications

For dryness, flaking and scaling following therapy of acute inflammation
Dermatitis of the hands ("Housewives' eczema")
In chronic atopic dermatitis
Pruritus senilis—pruritus hiemalis
In nummular dermatitis
In psoriasis
Xerosis or asteatosis
Sunburn
Diaper rash
Chafed or chapped skin
Ichthyosis

Caution: Lowila Emollient is partially occlusive. Therefore it should not be used until the skin is free of inflammation.

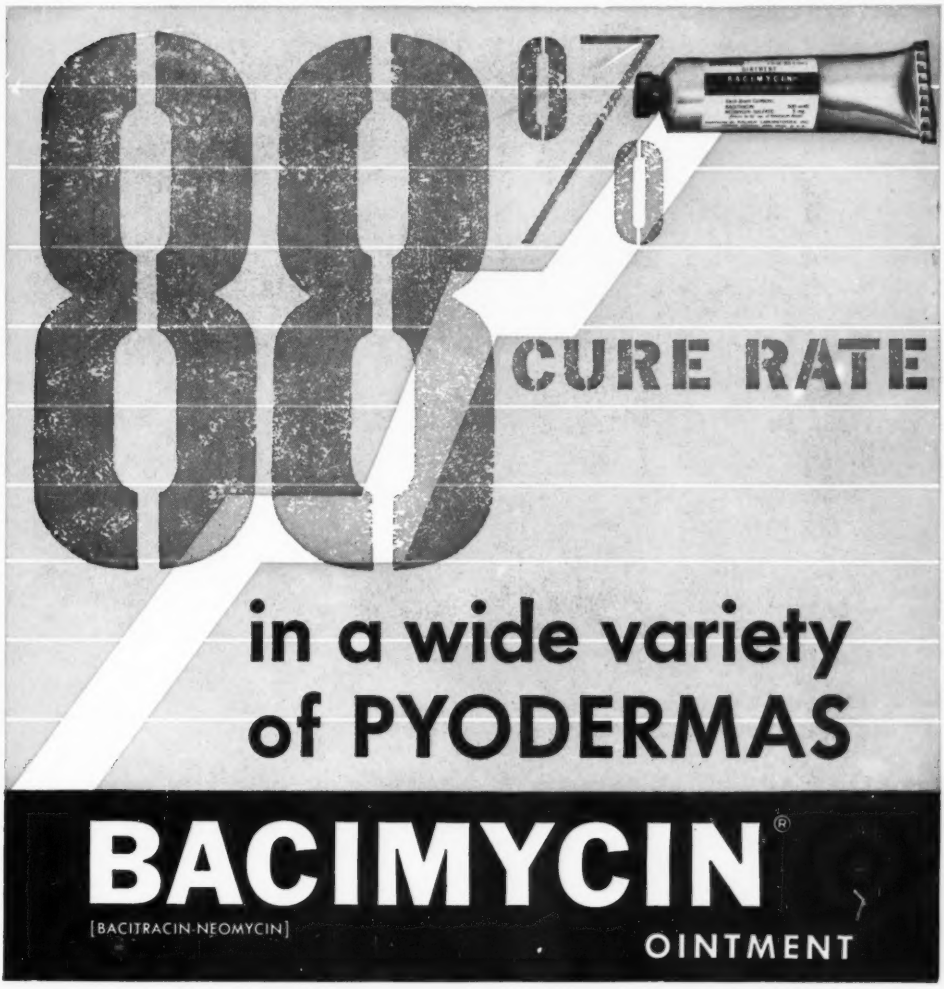
Application:

Gently rub a small amount into affected areas three or more times a day and at bedtime. Use particularly after exposure to water, soap and detergents.

Availability:

Lowila Emollient is supplied in 62.5 gram tubes.

WESTWOOD PHARMACEUTICALS
Division of Foster-Milburn Company
Buffalo 13, New York



a first choice for dual antibiotic therapy

In a recent study[1] of 53 patients with various types of pyodermas, the use of BACIMYCIN Ointment "... resulted in a cure rate of 88%...." Impetigo, infectious eczematoid dermatitis, atopic eczema, secondary infections superimposed on dermatitis venenata, and folliculitis were among the common skin infections that showed marked improvement with BACIMYCIN therapy.

BACIMYCIN rarely produces sensitization or primary irritation.

Supplied in ½ oz. tubes for prescriptions; in 100 gm. jars for hospital use; in ⅛ oz. tubes for ophthalmic use. Also supplied as BACIMYCIN with Hydrocortisone, ⅛ oz. tube with applicator tip.

Literature and samples on request.

1. Greenhouse, J. M., and Ryle, W. C.: A.M.A. Arch. Dermat. & Syph. 69:366 (March) 1954.

Walker **LABORATORIES, INC.** MOUNT VERNON, NEW YORK

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Chlamydospore Agar

Littman's Oxgall Agar

"It is considerably cheaper to use these bottles once and discard them than to pay the labor cost of reconditioning an equal number of test tubes."

...*Practical Medical Mycology*
By J. Walter Wilson, M.D. and Orda A. Plunkett, Ph.D.
Arch D & S 59:414, April 1949

A PREFERRED METHOD for the CULTURE of FUNGI as an OFFICE PROCEDURE

WRITE FOR INFORMATIVE LITERATURE

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